



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>



LANE MEDICAL LIBRARY STANFORD
L138 .J15 1980
A treatise on diphtheria / by A. Jacobl.



24503418663

29C

—PRESENTED TO—



By

The Society of the New York Hospital,
March, 1898.





•

•

•

YBA 881 3MA

COPYRIGHTED BY
WILLIAM WOOD & COMPANY
1880.

STEAM PRESS OF
H. O. A. INDUSTRIAL SCHOOL,
187 & 189 E. 76th St.

.138
516
1880

PREFACE.

My first paper on diphtheria, which appeared in the American Medical Times of August 11th and 18th, 1860, was based on several hundred cases. Since that remote period I have seen thousands, and published my experience several times. After some "Contributions" in the Journal of Obstetrics and Diseases of Women and Children, Feb., 1875, I collected and condensed my views in the second volume of C. Gerhardt's Handbuch der Kinderkrankheiten, 1877. Thus the present little book may be considered as an augmented edition of that monograph, with this exception, that it contains less literature and more therapeutics.

While thus admitting the claims of the practitioner as paramount, I was ever of opinion that a careful physician and therapist required the very latest and soundest results of exact scientific investigations as the foundation and safeguard of his practice. For the physician's science is no longer mere formula, and his art no routine. As the important questions of the identity and non-identity of "croup" and diphtheria, and of the parasitic or chemical nature of the diphtheritic poison are not yet

settled to the satisfaction of all, their discussion is still in order. I am bound to say, however, that anxious though I be for a possible future success of a parasitic theory of nosogenesis, I have not been able to change my attitude in regard to the prevalent bacteria doctrine of diphtheria. I cannot look upon the bacteria epidemic in the medical journals, particularly of Germany, with the hasty conclusions and gratuitous assumptions of scores of experimenters and writers, as anything but a calamity which, I trust, is but temporary. The safest verdict of the sober critic is still: "not proven." Fortunately, the non-acceptance of the bacteria doctrine does not at all interfere with the success of the rational practitioner.

The cases of diphtheria in every physician's practice are so numerous that, with very few exceptions, I shall abstain from detailing histories. What I hope to present in this little book is a condensed but tolerably extensive report on the present state of what is known about diphtheria, with my personal views on its pathology and treatment.

A. JACOBI, M.D.

NEW YORK, October 15th, 1880.

When the above was written and printed, I came into possession of Supplement No. 7 of the National Board of Health Bulletin. It contains a "research on the effects of inoculating the lower animals with diphtheritic exuda-

tion," by Drs. H. C. Wood and Henry F. Formad, with the signature, "University of Pennsylvania, July 1st, 1880." In thirty-two experiments, in which inoculation of diphtheritic matter was performed subcutaneously and in the mucous membrane of the mouth, the animals died in only six. The question naturally arises as to whether the few animals in which the inoculation was followed by death died of diphtheria or some other disease. Only in one case were there any exudations present in any organ which could give rise to the slightest suspicion that the animal died from diphtheria. It has been asserted by Oertel that animals which have been inoculated with diphtheritic material die with their internal organs infested with micrococci, and that the presence of these is characteristic of diphtheria. Wood and Formad have carefully examined the internal organs of the rabbits which died, as well as the blood of those which survived, and found no micrococci. In this their results are in complete accord with the very careful labors of Curtis and Satterthwaite.

The utmost care is necessary to prevent the entrance into the blood of bacteria from without. Thus they have cut the jugular veins of a rabbit and examining the blood at once, found it entirely free from bacteria. When, however, after the lapse of a few minutes the post-mortem was concluded and the heart opened, the blood therein contained possessed an abundance of these low organisms.

If the animals did not die of diphtheria, of what did they die? In every case the internal organs were tubercular.

But this condition was not due to inoculation with diphtheritic material only. Nine experiments were made with small masses of innocuous foreign material which was put under the skin. In five out of nine of these experiments tubercle was found after death; this large proportion apparently demonstrates that a simple local inflammation may in the rabbit act as a source of tubercular infection. Where diphtheritic matter was inoculated, inflammation was almost always induced at the seat of the lesion, with the formation of large lumps containing cheesy matter. These facts being so, it is a fair deduction that the tubercles were secondary to these inflammatory foci, and were therefore an indirect and not a direct result of the inoculation. Thus, diphtheritic membrane placed under the skin or in the muscles of rabbits may cause death in a few hours by the production of a blood poisoning which is not accompanied by any specific symptoms or lesions; or, after many days, by the development of a secondary tuberculosis.

Experiments prove that a pseudo-membranous tracheitis can be produced by placing the exudation matter in the trachea. Other experiments show that ammonia is able to produce in the cat and dog, as well as in the rabbit, a pseudo-membranous tracheitis. Prof. Oertel states that the membrane produced by cauterization of the trachea differs from diphtheritic membrane in containing no bacteria. What has led him to such an assertion cannot be comprehended. When the death occurred very quickly

bacteria and micrococci may have been less abundant in the traumatic membrane than in that taken from the throat of patients, but when the animal survived some days, and the bacteria had sufficient time to develop themselves, when, in other words, they were afforded as good opportunity of growth as in the natural disease, they were immensely abundant, in some cases seeming to make up a large part of the bulk of the membrane.

If it be possible to produce a fatal pseudo-membranous tracheitis by placing the diphtheritic membrane in the trachea, and not possible to cause septicæmia by inoculating other portions of the body with the same material, it would appear as though diphtheria might be originally a local disease with a subsequent septic poisoning. Experiments were therefore performed to determine whether any products of disease other than diphtheritic exudations are capable of causing pseudo-membranous tracheitis. In two of the ten experiments it was caused by the introduction of organic matter into the trachea. In both of the cases in which false membrane was produced, the injected material was pus; only four such experiments were made, so that the proportion of successful results is very large; much larger indeed than with true diphtheritic exudation in the experiments of Drs. Wood and Formad.

Trendelenburg found that not only ammonia, but also various other chemical irritants are capable of causing the formation of false membrane in the trachea; many years ago it was proven that tincture of cantharides will do the

same thing. It would seem, therefore, that in the trachea the formation of a pseudo-membrane is not the result of any peculiar or specific process, but simply of an intense inflammation, which may be produced by any irritant of sufficient power. This fact, certainly, is very suggestive in regard to the pathology of diphtheria.

A general view of the anatomical and clinical facts seems to indicate that the contagious material of diphtheria is really of the nature of a septic poison which is also locally very irritant to the mucous membrane; so that when brought in contact with the mucous membrane of the mouth and nose it produces an intense inflammation without absorption by a local action. Whilst absorption is not necessary for the production of the angina, it is very possible that the poison may act locally after absorption by being carried in the blood to the mucous membrane. Further, under this theory it is possible that the poison of diphtheria may cause an angina which will remain a purely local disorder, no absorption occurring, or a simply local tracheitis produced by exposure to cold or some other non-specific cause may produce the septic material when absorption will cause blood poisoning, the case ending as one of adynamic diphtheria. Some such an explanation as these here offered seems to reconcile the antagonistic opinions concerning the value of local treatment in diphtheria; because it is plain that the value of such treatment must largely depend upon whether the angina has or has not been preceded by absorption.

From the exanthemata diphtheria differs from the fact that one attack in no way protects against a second. It ranks rather with septic diseases which may recur indefinitely.

At present it seems altogether improbable that bacteria have any direct function in diphtheria, *i. e.*, that they enter the system as bacteria and develop as such in the system, and cause the symptoms. It is, however, possible that they may act upon the exudations of the trachea as the yeast plant acts upon sugar, and cause the production of a septic poison which differs from that of ordinary putrefaction, and bears such relations to the system as to, when absorbed, cause the systemic symptoms of diphtheria. Now, these bacteria may be always in the air, but not in sufficient quantities to cause tracheitis, but enough when lodged in the membrane to set up the peculiar fermentation; whilst during an epidemic they may be sufficiently numerous to incite an inflammation in a previously healthy throat.

The above extracts are reprinted almost literally from the remarkable paper of Drs. Wood and Formad, which is as conclusive as it is brief. Nothing equally excellent has been contributed to the literature of diphtheria for a long time. It appears to me that the mooted question of the essentiality of bacteria in regard to the nature and definition of diphtheria, to which many a page of this book had to be given up, is finally settled by them. While I regret the impossibility of quoting them at the proper

place and at proper length, I must not refrain from giving the greatest possible publicity to their conclusions, and expressing my satisfaction at being capable of sustaining my own position by their experiments, the skilful performance of which is surpassed only by their happy results.

A. JACOBI.

CONTENTS.

CHAPTER I.	History.....	1
CHAPTER II.	Etiology	27
CHAPTER III.	The Manner of Infection.....	51
CHAPTER IV.	Contagion and Incubation.....	57
CHAPTER V.	Symptoms.....	68
CHAPTER VI.	Anatomical Appearances.....	108
CHAPTER VII.	Diagnosis.....	134
CHAPTER VIII.	Prognosis.....	149
CHAPTER IX.	Treatment.....	154

A TREATISE ON DIPHTHERIA.

CHAPTER I.

HISTORY.

Diphtheria is a specific, infectious, and contagious disease characterized principally by epithelial changes in, and the exudation of fibrin on and into mucous membranes, the surface of wounds, and the rete Malpighii, thereby constituting the so-called pseudo-membrane. Under the names: *ulcus syriacum*, *ulcus ægyptiacum*, *garotillo*, *morbus suffocans*, *morbus suffocatorius*, *affectus suffocatorius*, *pestilentis gutturis affectio*, *pedancho maligna*, *angina maligna*, *angina passio*, *mal de gorge gangréneux*, *ulcère gangréneux*, *angina polyposa*, *angine couenneuse*, *synanche*, *croup*, *diphtheritis*, and *diphtheria*, the disease has been known and described at different periods by the writers of different nations. Aretæus, of Cappadocia, is notably the first of whom we have authentic proofs, if we except Asclepiades only, who is said to have performed laryngotomy. The description of the pharyngeal and laryngeal manifestations furnished us by the former, however, can leave no doubt in our minds that he knew diphtheria and recognized it. Galen tells us that the pseudo-membrane was gotten rid of by coughing when the respiratory passages were affected by the disease, and by hawking when the disease was in the pharynx. Cælius Aurelianus recognized diphtheria of the pharynx and larynx, as well

as the diphtheritic paralysis of the soft palate; it is to him we are indebted for the information that Asclepiades resorted to scarification of the tonsils, and even to laryngotomy. Aëtius, in the fifth century, advised against energetic local treatment and the forcible removal of the deposits before they were in a condition to fall off spontaneously.

In the literature of the middle ages, there are no proofs of the occurrence of diphtheria; still, some reports of gangrenous diseases, probably, have reference to it. Petrus Fosterus has given a fair description of an epidemic in Holland in the year 1557. Antonio Soglia, quoted by Chomel, describes an epidemic in Naples and Sicily (1563) which spread, in the following year, as far as Constantinople; Joannes Wierus, epidemics in Dantzic, Cologne, and Augsburg (1565); Ballonius (Baillou), in Paris (1576). In Spain, epidemics raged in the years 1583, 1587, 1591, 1596, 1600-1605, and 1613. Mercado (1608) tells of a child that had communicated the disease to its father by biting his finger. Cascalez advised gargles of solutions of alum and of sulphate of copper. Herrera (1515) described diphtheria of the skin and of wounds, and looked upon the pseudo-membrane he found after death as the essential characteristic of the disease. Heredia, in 1690, recognized the suffocative and asthenic forms, as well as the paralysis of the soft palate, of the pharynx, and of the limbs; he also called attention to the occurrence of relapses, which he attributed to the absorption of the morbid products, and endeavored to prevent by cauterization. In Portugal, too, there have been epidemics in early times. Barbosa, with whose work the world has become acquainted mainly through the meritorious labors of J. B. Ullersperger, describes one which occurred in 1626. Among the older writers on the subject we may mention Juan de Villareal, 1611, and Francesco de Figueiras in Lima, 1616. In Italy the first epidemic raged in 1618; René, Moreau, and Bartholinus detail a large number of literary proofs in support thereof;

it was characterized by its marked contagiousness and its depression of the vital powers, together with the weakness of the mental faculties left after an attack of the disease. The only autopsy, made in 1642, from a report by M. A. Severino, revealed the presence of pseudo-membrane in the larynx.* In Germany the disease was described by Wedel, in 1718. The epidemics observed by him were not very instructive, yet they sufficed to teach the importance of isolating the sick.

In the New England States diphtheria has appeared in the seventeenth century. Samuel Danforth, graduate of Harvard University, in 1643, had twelve children. The first died at the age of six months. The "next three being attacked by the 'malady of bladders in the windpipe,' in December, 1659, it pleased God to take them all away at once, even in one fortnight's time." This happened at Roxbury, Mass. (Biographical sketches of graduates of Harvard University, etc. By John Langdon Sibley, vol. I., Cambridge, 1873, p. 91.†)

John Josselyn, Gent., in "an account of two voyages to New England, made during the years 1638, 1663" (Boston, Wm. Veazie, 1865),‡ writes as follows (p. 141): "also they" (the English in New England) "are troubled with a disease in the mouth or throat, which hath proved mortal to some in a very short time, quinsies and impostumations of the almonds, with great distempers of cold." As Josselyn on his second voyage spent eight years in New England (principally in Maine), the foregoing must point to an epoch not later than 1671, and the manner in which he expresses himself shows that he does not speak of a recent invasion, but of a disease which had been common for

* See literature in Sanné, *Diphth.*, 1877, and Hirsch, *Hist. Geogr. Path.*, II.

† This important historical reference I owe to the watchfulness of Dr. Elsworth Eliot, New York.

‡ This book was published in 1674, and reprinted in 1833, by the Massachusetts Historical Society, and may be found in the third volume of the third series of their collections.

some time. I have no proofs of epidemics during the following decades, but the year 1735 marks the recurrence of epidemics. In a "Compendious History of New England" by Dr. Jedediah Morse, D.D., and Rev. Elijah Parish, Charlestown, 1804, we read with reference to the epidemic of 1735, that the throat became swollen and coated with white and ash-gray spots, an eruption appeared on the skin; great debility overcame the entire body, with a marked tendency to putridity. The disease first appeared in 1735, at Kingston, N. H. Wm. Douglass (The Practical History of a new epidemical Eruptive Military Fever with an angina ulcusculosa, which prevailed in Boston, New England, in the years 1735 and 1736) says of this epidemic: "It was first noticed in Kingston township, on the 20th of March, 1735, about fifty miles eastward of Boston. As this was an inland place of no considerable trade or importance, it was thought (incorrectly perhaps) to be of indigenous origin, and not of foreign importation. The first victim was a child who died in three days; and about a week after, three children were seized in another family four miles distant; and they also died on the third day. It continued spreading gradually, seizing here and there particular families, with that degree of violence that of the first forty cases none recovered. Some of the patients died of a sudden acute necrosis, or mortification; but most of them were carried off by a sympathetic affection of the fauces, neck, or air-passages; or, by an infiltration and tumefaction of the chops, and forepart of the neck which became so enlarged and turgid, as to bring upon a level all parts lying between the chin and sternum, occasioning a strangulation of the patient in a very short time. After a few weeks it spread from Kingston to the neighboring townships, but in a milder form. No reasons could be given for this greater malignity in Kingston, except, perhaps, the prevalence of damp places near large ponds, and fresh water, but sluggish streams, like in those localities which produce the rot in sheep. There may

also have been bad medical treatment. Its first recognized appearance in Boston was on the 20th of August, 1735, in a child . . . who had white specks in the throat, and a cutaneous efflorescence. A few more . . . were seized in like manner. Towards the end of September it appeared in several parts of the town of Boston, with more decided complaint of soreness of the throat. The tonsils were swelled and specked; the uvula was relaxed; there was slight fever, and an erysipelas or scarlet fever like efflorescence on the neck, chest, and extremities. The first alarming case was in the beginning of October, in a young man. He had lately arrived from Exeter, to the eastward of Boston, where his brother had died of the same illness. His symptoms were great prostration of strength, a single speck on one of his tonsils, and colliquative sweats. . . . It increased during the winter up to the second week in March, 1736; when it was at its height, there being twenty-four burials in all, during the week (instead of nine or ten). . . . The disease was so much milder in Boston than in some of the townships where it first prevailed that many could not be persuaded that it was the same disorder. . . . To the eastward of Boston, at times, one in three died, in other places one in four, and in scarce any towns, less than one in six; whereas in Boston not above one in thirty-five succumbed."

The disease cannot have remained so mild for any length of time in Boston, for on August 5th, 1740, the preface to the letter of I. Dickinson, A.M., dated at Cambridge, speaks of the "most malignant disease which had raged for a long time in the place where he lives, and which had commenced its fatal progress in these parts," and of the "fresh alarm by a return of that astonishing distemper among us." The letter was written in 1738, and published two years afterward, under the title of: "Observations on that terrible disease, vulgarly called the Throat Distemper, with advices as to the method of cure, in a letter to a friend. By I. Dickinson, A.M. Boston. Printed and sold by S.

Kneeland and T. Green, in Queen Street, over against the Prison. 1740."*

The clergyman to whose observations and industry we owe this short letter appears to have seen most of the forms of diphtheria. He describes cases which

1. "Begin with a shivering, a chill, or with stretching, or yawning, which is quickly succeeded with a sore throat, a tumefaction of the tonsils, uvula and epiglottis, and sometimes of the jaws, and even of the whole throat and neck. From the second to the fourth day there may be a miliary eruption, in some exactly resembling the measles, in others more like the scarlet fever, in others like the confluent small-pox." This form proved fatal but seldom.

2. "It frequently begins with a slight indisposition, much resembling an ordinary cold, with a listless habit, a slow and scarce discernible fever, some soreness of the throat and tumefaction of the tonsils; and perhaps a running of the nose, the countenance pale, and the eyes dull and heavy. Sometimes vehement sickness of the stomach, a perpetual vomiting; and sometimes by ejecting of black or rusty and fetid matter, having scales like bran mixed with it, which is a certain index of a fatal mortification. . . . When the lungs are thus affected, the patient is first afflicted with a dry, hollow cough, which is quickly succeeded with an extraordinary hoarseness and total loss of the voice, with the most distressing asthmatic symptoms and difficulty of breathing, under which the poor miserable creature struggles, until released by a perfect suffocation, or stoppage of the breath. . . . Comparatively few recovered. . . . Some expectorated incredible quantities of a tough whitish slough from their lungs. . . . I have seen large pieces of this crust, several inches long and near an inch broad, torn from the lungs by the vehemence of the cough."

* Reprinted by Stephen Wickes, A.M., M.D., on p. 87 to 99 of his excellent "History of Medicine in New Jersey, and of its Medical Men, from the settlement of the Province to A.D. 1800. Newark, N. J., 1879."

3. It sometimes appears in the form of an erysipelas, in some with all the terrible symptoms above described, in some with none of them.

4. In external ulcers all over the body.

5. In glandular swellings with favorable result only when they undergo suppuration, and lastly as sudden orthopnœa, which proves fatal in from one to three days. It shows its peculiar character by the presence of the "crustula in the throat, which determines it to be a sprout from the same root with the symptoms described above."

The treatment of the Reverend is a singularly rational one; moderate temperature, disinfectant applications, warm poultices to swollen glands, but moderate purgation, and mild stimulants, all of them in the compound mixtures of his time, form his armamentarium. He adds that there is no security against a second attack; he has seen it four times in a patient in one year, the last proving fatal, and known numbers that have passed through it in the eruptive form in the summer season, "that have died with it the succeeding fall or winter, though I have never seen any upon whom the eruptions could be brought out more than once."

According to Dr. Cadwalader Colden, in a letter written 1753 to Dr. Fothergill (printed in vol. 1st of the London Med. Observations and Inquiries, pp. 211-225), the "throat distemper" spread from Kingston gradually westward, so that it did not reach the Hudson river till near two years afterwards. It continued some time on the east side of the river, before it passed to the west, and appeared first in those places to which the people of New England chiefly resorted for trade, and in places through which they travelled. It continued to move westerly, till it probably spread over all the British Colonies on the continent. Dr. Colden's description resembles that of Dr. Douglas. A few of the points emphasized by him are the following. Though the disease was evidently propagated by infection, yet children and young people were only subject to it, with

the exception of a few. A very few aged people who were taken died. It did not spread equally to all places that were proportionately exposed to the infection. The poorer sort of people were more liable to it, and they who lived on low and wet grounds and on poor scorbutic diet. In some places, only a few persons or families were seized; while in others, all escaped. In some families it passed like a plague through all their children; in others, only one or two were seized. Some were attacked at great distances. Some had it mildly, while others in the same place and at the same time had it most violently. . . . "Many have not been confined to their beds, but have walked about the room, till within an hour or two of their death. And the complaint has often appeared no way dangerous, at first, to the attendants, till the sick were almost in the last agonies, though the patients themselves are generally dejected and apprehensive. . . . When the surfaces of the tonsils, after the sloughs were cast off, appeared of a very fiery-red color, there was some, or even great danger; but when they were covered with a black crust it was often a fatal omen, as also where hemorrhages followed any slight scratch. . . . When the disease first appeared, it was treated in the usual way for a common angina, and no plague was more destructive. . . . The orifices made by the lancet in bleeding, and the adjacent parts were apt to become diseased. So likewise the places where blisters were applied. The ichor which issued from them corroded the parts upon which it flowed, and even slight scratches became as it were mortified. . . . A girl about ten years of age, while the throat distemper was prevailing, had sores on her private parts like those on the tonsils of others, but no symptom of the disorder appeared in her throat."

The epidemic does not appear to have abated much after this time. For, in his first letter to Mr. Hugh Gainé, of New York, dated Jamaica (Long Island), Oct. 28th, 1769 (reprinted in the "Medical Repository," Vol. V.,

New York, 1802, p. 97), Dr. Jacob Ogden refers to the "distressed condition of the people in Boston and Oxford, occasioned by the fatal effects of the malignant sore-throat distemper." He also refers to the prevalence of the disease in his neighborhood. The symptomatology contained in his second letter, dated Jamaica, Sept. 14th, 1774, contains, briefly, a recapitulation of the excellent description, mainly of the milder forms, as given by Douglas, while the first is given up to treatment alone. Calomel and a compound mixture containing Seneka (*Polygala s.*) are highly recommended. Seneka was, credit not being given, claimed as the sufficient remedy in diphtheria "without any other medicine—the use of mercury may, however, sometimes, as in the symptomatic kind, be necessary"—by John Archer, Jr., in "an inaugural dissertation on cynanche trachealis, commonly called croup or hives," Philadelphia, 1798, p. 46, and "on the use of the Radix Seneka in the cure of croup. Letter to B. S. Barton, M.D." *Med. Repos.*, I., p. 120, 1798; and II., 1799, p. 27. In connection with the above, a few sentences will be of interest as showing the tendency to disinfecting treatment on the part of the old practitioner. "Detergent gargarisms, or injections, are of service when the patient is of an age capable of using them. The air of the room where the sick lie might also with advantage be continually impregnated with the steam of vinegar. Sea-coal might be burnt in the winter instead of other fuel; and, in the summer, the room might be fumigated with myrrh, rosin, and sulphur, strewed on burning coal often in a day. These fumes, if diligently persisted in, would, it is more than probable, not only much benefit the sick, but be the best means of preventing the infection from spreading in the family" (*Med. Repos.*, V., 1802, p. 103).

If we disregard the dubious reports of Le Cat concerning epidemics in Rouen in 1736 and 1737, we must consider them to have first occurred in France in 1745, having begun in Paris (Astruc, Malouin, Bouillaud, Cho-

mel, du Hamel, Boucher, Navier), and later invaded the provinces (Rolin, Grandvilliers). Chomel has given us an accurate description of paralysis of the soft palate, and reports a case of diphtheritic strabismus. The English epidemic, described by Starr, occurred in the years 1744 to 1748; that by Huxham, in Plymouth, in 1751 to 1753. The first Swiss epidemic happened in 1752, the Dutch in 1747 (Zaff), the Swedish in 1755 (Berg).

During the subsequent part of the eighteenth century, but two writers are worthy of especial notice, Home, a Scotchman, 1765; and Samuel Bard, an American, 1771.

Home deserves credit for having distinctly drawn the line between the pseudo-membranous and the gangrenous affections, and also for having endeavored, to his utmost, to convince his colleagues that croup and angina maligna were two distinct diseases, notwithstanding all that had been uttered since the time of Aretæus to prove their identity. No matter in what way he tried to explain the false membranes—he looked upon it as an aggregation of mucus—he found it, and gave an accurate description thereof. He sought for it exclusively in the respiratory tract, and disregarded any connection between it and the false membrane found in the pharynx.

Bard's experience was very extensive; he saw membranous pharyngitis, membranous laryngitis, and membranous pharyngo-laryngitis; he speaks of the membrane affecting the skin, of paralysis of the muscles of deglutition, and of the larynx, and likewise of paralysis of the lower extremities, as sequelæ. He looked upon the morbid process as the same, whichever were the mucous membranes attacked, and made a distinction only according to the localization of the disease. The influence which he might have exercised in shaping the professional opinion on the nature of the disease did not make itself felt, partly because of the classical modesty of the New York physician, and partly because of his remoteness from the centres of European learning.

"Bard's article is among the calmest, wisest, and most accurate that has ever been written on diphtheria, both before and since his time."* While his style is classical in its simplicity, his observation is astonishingly correct, and his conclusions as to the actual identity of all the diphtheritic processes in the most various clinical symptoms unimpeachable this very day. His description of the various forms of pharyngeal diphtheria is painfully good, his observations on cutaneous diphtheria very accurate, his few dissections well recorded, particularly when he speaks of tracheal and tracheo-laryngeal diphtheria, and his historical reviews very judicious indeed. "Upon the whole, I am led to conclude that the morbus strangulatorius of the Italians, the croup of Dr. Home, the malignant ulcerous sore-throat of Huxham and Fothergill, and the disease I have described, and that first described by Dr. Douglas, of Boston, however they may differ in symptoms, do all bear an essential affinity and relation to each other; or are apt to run into each other, and, in fact, arise from the same leaven. The disease I have described appeared evidently to be of an infectious nature, and being drawn in by the breath of a healthy child, irritated the glands of the throat and wind-pipe. The infection did not seem to depend so much on any prevailing disposition of the air, as upon effluvia received from the breath of infected persons. This will account why the disorder sometimes went through a whole family, and yet did not affect the next-door neighbors. Here we learn a useful lesson, viz.: to remove young children as soon as any one of them is taken with the disease, by which many lives have been saved and may again be preserved."

"A letter from Peter Middleton, M.D., to Mr. Richard Bayley on the croup," dated New York, Nov. 30th, 1780, which had become absolutely rare for some time, and was reprinted, therefore, in the *Med. Repos.*, Third Hexade,

* John C. Peters in *West Virginia Med. Stud.*, Aug., 1876.

Vol. II., N. Y., 1811, p. 347, contains Dr. Middleton's views on the nature of the prevailing illness. He was a Scotchman, who emigrated to the colony in 1752, and coincided with Home's views on croup. He insists upon having met with local membranous croup only, though he does not "presume to say that it never is complicated with the malignant sore-throat," and asserts to have been very successful with jugular venesections, blisters over the throat, and evacuants.

Dr. Richard Bayley's letter to Dr. William Hunter, of London, written and published about the year 1781, shared the fate of Dr. Middleton's pamphlet. It got scarce, and at last lost, so that only a part of it could be reproduced in the Med. Repos. of 1809, Second Hexade, Vol. VI., p. 331, while the then lost part was supplied in that of 1811, Third Hexade, Vol. II., p. 345. While Dr. Middleton denies having seen the throat distemper with his cases of croup, Dr. Bayley describes the same class of cases with the exact symptoms of pharyngeal diphtheria. The main symptoms were, however, with him also those of local obstruction and suffocation, of "angina trachealis." He considers it as "an inflammatory disease, the treatment of which must vary in every degree according to its violence; and though the common antiphlogistic treatment will in some cases relieve, if early applied, yet the most desperate may yield to repeated bleedings *ad deliquium* from the jugulars, the free use of tartar emetic and other evacuants, with a large blister covering the larynx and aspera arteria, while the mucus filling up the ramifications of the bronchia may be emptied by the action of vomiting."

Jurine, in his prize essay of 1807, goes no further than to deny the gangrenous nature of angina maligna, and to emphasize the frequent complication of membranous croup with membranous pharyngitis. It was reserved for Bretonneau to enforce attention to the ideas of Bard, by asserting (though he did not mention either his monograph or its French translation of 1811) the identity of angina

maligna, or by whatever other title it may be known, with membranous laryngitis, and, by inaugurating his theory with a new name for the disease, to perpetuate the views expressed therein. First and foremost, he called attention to the continuity of the membrane (according to him, composed of coagulated mucus and fibrin) of the nose, pharynx, and respiratory tract, its identity with certain morbid conditions of the skin, and promulgated the theory that diphtheria is a specific disease, an affection *sui generis*, to be distinctly separated from a catarrhal, as well as from a scarlatinous inflammation.

The modern history* of diphtheria may be dated from June 26th, 1821, when Bretonneau read his first essay on that subject before the French Academy of Medicine, and gave to the disease the name it now bears. His second and third (Nov. 25th) papers belong to the same year, his fourth being read in March, 1826, his fifth appearing in the Archives Gén. of January and September, 1855. It was only in 1826 that the material, previously gathered, was summed up in his celebrated monograph. Before this time, however, the separate essays had received prominence from the reports and commentaries of Guersant, who laid particular stress on the statement that diphtheria is a non-gangrenous affection, identical, and even synchronous with croup in the majority of epidemics. Since that epoch, the literature on the subject has assumed enormous proportions. A short résumé of the writings in reference thereto that have since appeared is all that can reasonably be offered, but it is interesting to note that many important questions bearing on the etiology, pathology, and treatment of the disease were then, as they still remain, mooted points.

Bourgeoise, in a paper read before the Academy, and Brunet, already in 1823, expressed their belief in the contagious character of the disease. Desruelles (1824) sees a

* Compare John Chatto, in "Memoirs on Diphtheria," Sydenh. Soc.

diagnostic point between the sporadic and the epidemic forms in the participation of the brain in the latter. Louis referred a number of cases of croup in adults to pharyngeal diphtheria as their source. Gendron (1825) recommends lunar caustic and scarification of the tonsils as prophylactic and curative agents. Mackenzie considers that croup has its origin in the fauces, and urges the employment of lunar caustic. Billard (1826) denies the specific character of diphtheritic inflammation. Hamilton describes cases that terminated in suppuration, and which he therefore distinguishes from Bretonneau's cases. He describes two modes of termination of the disease, one in croup, the other in a state of debility arising from the effect of the absorbed secretion on the respiratory nerves. Pretty looks upon those cases of croup that have their original seat in the tonsils, as contagious. Conolly gives us a description of the disease similar to that of Bretonneau, and recommends calomel. Bland (1827) explains the difference between croup and diphtheria. Deslandes declares them to be identical. Bretonneau publishes a work in which he compares diphtheria with scarlatina anginosa, and recommends the use of alum. Emmangard is the first one of the "physiological" school who, likening diphtheria to typhoid, and claiming its origin in a malarial infection, calls it "angina gastro-enterica." Blanquin (1828), following him, recommends bleeding, by which he lost only fourteen cases out of three hundred. On the other hand, Belden recommends lunar caustic, Gendron lunar caustic, antiphlogistic treatment and scarification, and Lormel antiphlogistic treatment and counter-irritation. Abercrombie is in favor of distinguishing diphtheria from croup, but reports a number of cases of diphtheria of the pharynx that terminated fatally by stenosis of the larynx. Ribes, who encountered the disease in nine members of a single family, asserts that croup rarely occurred without a preceding diphtheria, in his experience; he advises an examination of the throats of apparently healthy individuals.

Fuchs relates the history of epidemics of angina maligna, and declares croup to be a genuine angina maligna trachealis, which only does not run through all the stages. Broussais opposes the identity of croup and diphtheria (1829), and gives a report of cures by means of antiphlogistic regimen and laryngotomy. Diphtheria and gangrenous angina are synonymous with him. Gendron expresses a belief in the identity of diphtheria and gangrenous angina, and advises antiphlogistics and lunar caustic, opposing the use of muriatic acid. Roche considers the membrane rather of hemorrhagic than of inflammatory origin, and consisting of discolored fibrin. Menon employs sinapisms, cathartics and antimony, but no topical applications. About the same time, Trousseau is endeavoring to clearly establish the diagnostic points between diphtheria and scarlatinous angina. Shortly after (1830), he reports cases of diphtheria having their origin in artificial blistering-wounds, and of diphtheria of the skin giving rise to throat affections, and diphtheria of the throat followed by skin disease. Regnier declares antiphlogistic treatment useless. Archambault-Reverdy protests against the abuse of local treatment. Velpeau, as also Girouard, two years later, suggests alum and lunar caustic. In 1831, we find Broussais inclining to the "gastro-enteric" view, and opposing Bretonneau's theory of the nature of the disease; while Gendron has thrown down the gauntlet in favor of its being contagious. J. F. Hoffman cites a severe case that ultimately recovered with consecutive paralysis of certain cranial nerves. Cheyne (1833) makes a stand against the "confounding of croup and cynanche maligna under the name of diphtheritis." Gendron reports two fatal cases after tracheotomy, and recommends lunar caustic. Lemer cier favors the use of lunar caustic and bleeding. Cotlineau complains of the too infrequent employment of internal remedies. Richard (1834) advocates antiphlogistics and cauterization. Bourgeois witnessed an epidemic succeeding mumps, and found

muriatic acid, locally applied, of great service. Fricout and Burley (1836) declare their belief in the contagiousness of the disease. Bouillaud attacks the theory of its specific character on the ground that abstraction of blood produced favorable results. Stokes makes a distinction between primary and secondary croup, according to the original seat of the affection (1837). Baumgartner (1838), as also Bouchut, many years after, recommends partial removal of the tonsils, and Ruppius, bleeding and calomel; Bretonneau (1839), caustics; Geddings, of Charleston, S. C., lunar caustic and muriatic acid; Benson (1840), calomel internally, lunar caustic and mineral acids locally; Duplan (1841), bleeding and emetics. During the same year, Kessler advocates the view of its contagious nature, and Rilliet and Barthez adduce evidence of the occurrence of ulceration and gangrene in the course of the disease. Taupin, like Ribes, enjoins a methodical examination of the throat of every patient, during the prevalence of an epidemic of diphtheria, whatsoever be the disease from which the child suffers. Boudet (1842) opposes Bretonneau's hypothesis that croup is a descending diphtheria, and holds to the identity of diphtheria and gangrenous angina. In this contest, Durand (1843) also takes sides against Bretonneau, and lays particular stress on the point that the diphtheritic patient succumbs rather from the severity of the constitutional symptoms than from suffocation. Rilliet and Barthez, on the other hand, rally to the support of the attacked master, asserting that the usual form of croup, and that resulting from a declining diphtheritis, are one and the same, while they claim that diphtheritis and gangrenous angina are distinct affections. Becquerel advises local cauterization and tonic treatment; Moland (1845), lunar caustic; Daviot (1846), lunar caustic, and in children over ten years, depletion; Hein (1849), calomel and lunar caustic; Beck (1850), emetics, while he opposes caustics; Bourgeois, calomel and lunar caustic; Brown, mercury and lunar caustic;

Welsch, a strong solution of nitrate of silver, which he prefers to alum, sulphate of copper, and muriatic acid.

Meanwhile the strife regarding the nature of the disease continued. Guersant and Blache (1844) describe the "stomatite couenneuse" (noma, stomacace, according to them, the rarest kind of gangrenous angina) as a form of Bretonneau's diphtheritis, and Landsberg raises the question whether a nerve inflammation, present in a certain case, was to be looked upon as an accidental or an essential feature of the disease, and finally comes to the conclusion, with Schönlein, that it was a neurophlogosis dependent on the disease. Bouisson (1847) reports a case of diphtheritic conjunctivitis resulting in loss of the eye. Robert publishes his observations on diphtheria of the skin and of wounds, which he attributes to an atmospheric contamination in crowded wards of hospitals, and looks upon it, with Delpech and Eisenmann, as a form of hospital gangrene. Virchow, in the same year, distinguished the catarrhal, croupous, and diphtheritic varieties of the disease. The ensuing years are especially rich in suggestions as to the mode of treating diphtheria. In France, the alkaline treatment leads the van; Baron (1851) employing Vichy water; Lemaire (1853), bicarbonate of soda. Daga (1854) urges the topical application of concentrated muriatic acid. Meanwhile reports of paralysis of the soft palate after diphtheria came from Morisseau, from Trousseau and Lasegue, and lastly (1854-'59) from Maingault. The subject of diphtheritic conjunctivitis was studied by A. v. Graefe (1854), who encountered the disease as a complication of diphtheria of the pharynx, nose, and skin, and hence considered it a part of the general disease rather than an independent local affection. Diphtheria, in its effects on the system, had at the same time been investigated by Trousseau, who sums up with the statement that the principal source of danger lies in the invasion of the larynx, and that the large majority of cases of croup began as a diphtheria of the pharynx, but that, even without

the occurrence of a laryngeal localization, many cases terminate fatally owing to adynamia.

Outside of France, too, the subject had attracted attention. West, who had never seen the disease occur primarily, describes diphtheria appearing as a complication of measles. Wunderlich and Bamberger (1855) enrich the records with their wonted zeal. The latter divides the inflammations of the mouth and pharynx into the catarrhal and croupous forms, and considers croup and diphtheria subdivisions of the latter form, differing only in degree. Meanwhile the French were almost exclusively engaged on the treatment. Bretonneau, while expressing a few remarks on the contagiousness of diphtheria, urges the employment of lunar caustic both as a remedial and as a prophylactic agent; similarly Latour and Ferrand. Marchal de Calvi thinks that the duration of the disease is thereby lengthened, and dwells on the favorable effects of the alkaline treatment, against which Latour and Marcuel express themselves. Valentine calls to his aid the actual cautery; A. Smith and Isambert (1856), Roux and Woodward (1857) having recourse to the chlorate of potassium, which, in the hands of the last-mentioned, would seem, however, to have been only serviceable in mild cases. Applications of tincture of iodine are recommended by Lecointe, Perron, and Boinet, and of bromine and bromide of potassium by Zanam (1856). Popo expresses himself in favor of muriatic acid and opposed to the use of alkalies; Gigot, in favor of alkalies and against cauterization (1857). The paralysis of the muscles of deglutition is discussed by Dehaenne, who had contracted the disease, and the paralysis of other muscles by Faure. A case of diphtheria of the tonsils, nipples, and vagina, in a woman recently confined, followed by infection of the new-born, and the death of both, is reported by Mahieux; and cases of diphtheritic conjunctivitis by Grichard, Warlomont, and Testelin. The same year, Isambert published a work in which he divided the diphtheritic affections into three forms, viz.: angine

couenneuse, scarlatinous angina, and diphtheritic angina. The last-mentioned is further subdivided into a croupous-diphtheritic angina, in which croup of the larynx plays an important part, and into that form in which death results from adynamia; in the latter form there is a marked swelling of the lymphatic glands. Apparently, at this time, the epidemic in Paris underwent a considerable change, for the croupous form does not occur by far as frequently as Bretonneau had asserted, and croup of the larynx without a preceding diphtheria of the pharynx was observed more often than he would lead us to believe.

Thenceforward the literature on this subject became unlimited, and the year 1858 saw an epidemic of contributions thereto. Beale calls his microscope into requisition, and finds no parasites. Laycock endeavors to trace the disease to the presence of the *oidium albicans*. Welks finds the same parasite in other affections. Cammack declares the diphtheritic membrane to be herpetic. Féron calls Bretonneau's mild form of the disease an herpetic angina, accompanied by a formation of false membrane; Gabler looks upon the same form as herpetic in character, and frequently accompanied by herpes labialis. Bouchut declaims against the identity of diphtheria, croup, and gangrene. Condie describes the disease as occurring with scarlatina. Litchfield claims that it is a concealed scarlatina, and Hillier (1859) that it has some connection with scarlatina. Millard cites one case in the course of which gangrene occurred, and another in which skin, mouth, pharynx, respiratory passages, œsophagus, and vulva were affected at the same time. Harley vainly endeavored to inoculate the disease in animals. Stephens declares the disease to be infectious. Sanderson looks upon it as identical with the angina maligna of the aged. Farr considered the exhalations from sewers an important etiological factor. Sellerier, Kingsford, and Harley (1859) report paralyzes as sequelæ. Mangin speaks of a specific eruption; Ward, of an accompanying purpura. Bouchut

and Empis remarked the frequent presence and corresponding danger of albuminuria; so did Wade. Maugin calls attention to the fact that, when present in diphtheria, it occurs early, whereas in scarlatina it is seen during the period of desquamation, and is not of frequent occurrence even then. Gull gives an account of cases in which death resulted from asthenia, and speaks of a nerve lesion which he attributes to the severity of the local inflammation. Hildige describes diphtheritic conjunctivitis as seen in Græfe's practice, and looks upon it as contagious. Magne denies its contagious or infectious character. Mackenzie, while probably having seen false membrane appear on the conjunctiva when in a state of inflammation, yet refuses to recognize diphtheritic conjunctivitis as a distinct disease. Reports likewise come from North and South America. According to Boston Med. and Surg. Journ., Vol. LIX., 1858, the epidemic in Providence, R. I., had a similar course to that in England; that in Albany and Troy, described by Dr. Willard (calomel treatment), being very malignant, and in New York, described by me, 1860, moderately severe. Odriozola, in a report from Lima, testifies to the immunity enjoyed by the blacks.

Therapeutical contributions are not less numerous. Iron, generally in the form of the chloride, is recommended by Barry (who also employs chlorate of potass., counter-irritation, and cauterization with nitrate of silver), Fourgeaud, in San Francisco, Gigot and Jodin, Heslop (together with cauterization with muriatic acid); Houghton (the same, and in addition tonic diet); Kingsford (tonics, nutritive enemata, chlorate of potass.); and Stiles (together with the topical employment of concentrated saline solutions). Bryan advises the use of guaiac internally and salt topically; Cazin, lemon-juice; Duché, sulphur; Gay, tracheotomy; Lambden, chlorate of potassium with muriatic acid, and cauterization with nitrate of potassium; Bulley warning against the indiscriminate employment of the latter; McDonald, Bass' ale and quinia, with

the local application of muriatic acid; Bouchut, removal of the affected portion of the tonsils, and internally glycerin, by which method of treatment he anticipates, with his usual enthusiasm, a prevention of croup of the larynx, and that the wound left after tonsillotomy will not become diphtheritic. Soon after he reports a case of croup in which restoration of the voice followed the introduction of a tube into the larynx. Borland publishes a fatal case of croup arising after amputation of the tonsils.

We shall more than once be obliged to refer back to the early history of the pathology and therapeutics of diphtheria. The former is in part a new creation, the latter greatly improved. For, however greatly the value of disinfecting agents, as the sulphites, carbolic and salicylic acids, administered internally, may be over-estimated, yet certain indications for their employment will ever remain, and new fields of action be opened to them. In the matter of local disinfection, a vast region of usefulness and benefit is at their disposal. I shall, however, not discuss these points here, as I propose dwelling on them in connection with the subject of treatment.

I also refrain from giving a detailed history of diphtheria for the past twenty years. It would be a monotonous and exceedingly ungrateful task. A great part of that history is but a repetition of what has been previously observed and reported. Fortunately, however, the ablest minds were engaged on the same topic. It is mainly the German literature which became, however, enriched by the results of investigation in pathology, rather than by the further addition to the already enormous category of remedial agents. To Virchow we are indebted for recognizing different forms of diphtheritic invasion, which, according to the depth of their seat, are deserving of different terms. The genuinely diphtheritic form extends into the mucous membrane, the so-called croupous lies on the surface thereof. In the former, the exudation occurs into the tissues of the mucous membrane, which thereby becomes de-

stroyed; in the latter, the mucous membrane remains intact, and is at the utmost deprived of its epithelium by the stripping off of the pseudo-membrane. This difference, between death of the tissue on one hand, and an exudation of fibrin superficially on the other, soon came to be believed in, however, as essential and vital; even the best text-books willingly looked upon diphtheria and croup as heterogeneous, though Virchow himself never committed himself to such heterogeneity, but only to divers modes of development.* E. Wagner, however, sought for evidence to prove that the two diseases were identical in character, and were only apparently distinct, owing to the difference in localization and, let us add, in clinical symptoms. He found the explanation of the diphtheritic process in a rapid granular degeneration and an equally rapid reproduction and renewed decomposition of epithelial cells. While Wagner attached the greatest importance to the local affection, Buhl endeavored to explain the latter by presuming a constitutional disease which produces lesions in different parts of the body, after the manner of the eruptive fevers. According to the latter view, the general infection would be looked upon as the essential feature, and the granular metamorphosis of the submucous connective tissue, which, by compression of the blood-vessels, leads to death of the tissue, as the result of the constitutional disorder. In contradistinction to the view that diphtheria is a general disorder with localized lesions, arose that which founds all miasmatic and infectious diseases on the presence of living organisms. Beginning with Salisbury,

* The American profession has been greatly influenced by two text-books, those of Niemeyer and of Vogel, in regard to this supposed difference between croup and diphtheria. To say that the only difference between two forms of disease, which unfortunately had different names, consists in the fact that the morbid process in the one is found on the surface; in the other, a little below the surface—while it is admitted that the histological condition is the same—speaks for an exaggerated tendency to classify and subdivide. It is just these chapters on croup and diphtheria in the very same text-books which leave more to desire than any other.

who, with marvellous rapidity, recorded hosts of microscopical parasites, to which he as quickly assigned names, and not ending with Hallier, the search for minute organisms was pursued with excessive assiduity.

So agreeable and tempting did it appear to unveil with one supreme effort the mystery attaching to miasmatic and contagious affections that literature became flooded with "communications" and "preliminary remarks." At the same time, however, a series of earnest and conscientious investigations were recorded. Hüter, Tommasi, and Oertel, the first rather enthusiastically, the latter more coolly, prove as the results of their observations and experiments that diphtheritic membrane, the blood, and certain of the tissues are the abode, in diphtheria, of bacteria, which, in their opinion, constitute the etiological factors of the disease. The investigations of Trendelenburg, Nassiloff, Recklinghausen, Waldeyer, Eberth, Klebs, and many others have amply enriched the literature bearing on this theory. According to it, diphtheria is an exclusively parasitic disease resulting from local infection, the bacteria being either the essential morbid element, as is held by some, or the carriers of the diphtheritic poison, according to the view of others. Fortunately this theory, so agreeable in itself, and apparently consistent with the exact and mechanical phenomena of nature, has received some wholesome check by the labors of Hiller, Curtis and Satterthwaite, Billroth, and others. Above all, be the bacteria a frequent, nay even a constant accompaniment of accidental wound diseases, and the vehicles of septic poison, we may still deny them an independent morbid power. In his great work on coccobacteria, Billroth asserts that even in pyæmia and septicæmia the existence of bacteria in the blood cannot be verified, and Charlton Bastian denies that the poison of infectious diseases is endowed with vital powers. This subject, however, will claim our attention in another chapter of this book. Whether the present disharmony in regard to the bacteria

question will tend to a satisfactory interpretation of the diphtheria problem in the sense of the exclusive and radical parasitists can hardly as yet be answered in the affirmative. I am inclined to deny it. At all events, it does not appear safe to go any further than Billroth does in his remarks on some forms of diphtheria (Allg. Chir. Pathol. u. Ther., 8th Ed., 381).

"In the pulpos mass of muco-salivary diphtheria (extensive diphtheritic destruction of the cheeks, etc., after operations on the jaws), and in the urinary diphtheria, micrococci and streptococci can always be found; they are met with as constantly in the secretions about the teeth and in the coating of the tongue, as well as in every specimen of ammoniacal urine, but appear to multiply with marvellous rapidity in the above-mentioned pulpos mass. The contagion peculiar to this mass could not as yet be separated from the micrococci, and the latter may, therefore, be presumed to possess, or be connected with, the contagious zymotic element; it has *not yet been proven that every micrococcus, whatever its source, may originate this element, but many observations demonstrate that these organisms may easily absorb certain contagious substances and hence act as carriers of contagion and ferments.* For example, if the cornea of a rabbit be inoculated with fluids containing the micrococcus, the coccus will fructify, as demonstrated by the interesting experiments of Nassiloff, Eberth, Leber, Stromeyer, Dol-schenkow, Orth, Frisch, and others; in some cases (where it is not accompanied by harmful substances) its action is principally an irritant one, separating the lamellæ of the cornea, so that the little coccus-colony is by degrees enclosed by a collection of pus and ultimately expelled with it; in other cases again (where the inoculated material is possessed of very deleterious qualities), the entire cornea becomes gangrenous in twenty-four hours, *although the coccus-formation have barely reached the degree of development that characterized the condition previously described.*

Finally there are cases in which the small coccus proliferation *excites no reaction* in the cornea, but soon disappears without leaving any traces behind; in fact, this is the rule where inoculations are undertaken on the cornea of dogs. From this it may be inferred that the *intensity of the inflammatory reaction produced by such contagious elements and the mode of its production are not dependent on the coccus-formation as such, but on whether the elements which it brings with it are deleterious or otherwise.*

SUMMARY.

Aretæus is the first whose description of diphtheria has reached us.

Asclepiades practised scarification of the tonsils and laryngotomy.

Cælius Aurelianus recognized diphtheria of the pharynx and larynx and the diphtheritic paralysis of the soft palate.

Frequent epidemics are known to have taken place in the second half of the sixteenth century over the larger part of Europe.

Diphtheria of the skin and of wounds was described by Herrera in 1515.

Communication of diphtheria through a wound in the finger is reported by Mercado in 1608.

An autopsy was made in 1642, and membrane found in the larynx.

The suffocative, asthenic, and paralytic forms of diphtheria were described by Heredia in 1690.

The first cases known in America occurred in Roxbury, Mass., in December, 1659. About that time, and mainly about 1671, the disease was very prevalent. It recurred, 1735, in New England, and never disappeared for any length of time until the beginning of this century. The main writers during this period are Douglas, I. Dickinson, Cadwalader Colden, Samuel Bard, Jacob Ogden, John Archer, Peter Middleton, Richard Bailey.

Samuel Bard proved the identity of all forms of diph-

theria, cutaneous, pharyngeal, nasal, laryngeal, tracheal. So did Bretonneau fifty years later, and Trousseau, Louis, x Rilliet and Barthey, and all the great clinicians.

Contagiousness was never doubted, but mainly sustained by Bourgeoise in 1823.

Virchow discriminates the catarrhal, croupous, and necrobiotic forms, 1847.

Graefe describes diphtheria on conjunctiva and cornea in 1854.

The main objects of the scientific literature of the subject in the last twenty years have been the microscopical histology and etiology, besides the reports of cases, epidemics, and therapeutics.

CHAPTER II.

ETIOLOGY.

Diphtheria is pre-eminently a disease of early life; in this respect it is said to differ from the genuine fibrinous bronchitis, which by some is held, an absolutely different disease and stated to occur but rarely in children. But even this statement is probably incorrect. In the single spring of 1879, I have met with four cases of fibrinous bronchitis in children under three years of age. The number of cases of diphtheria in adult life is not very large, while in old age it is very small. Of 501 deaths in Vienna in 1868, only one had reached the age of sixty-two; of more than 250 cases in which I performed tracheotomy—beside a young physician in New York, whom I saw during his illness, and on whom tracheotomy was performed by a colleague at his dictation, though against indication—but two were over thirteen years old. One of them was a male of fifty-five years, with ossified tracheal cartilages, who perished from gangrenous and septic diphtheria and pneumonia thirteen days after the operation. The other was a lady of thirty-odd years, rather stout and anæmic, but enjoying tolerable health withal, who had given birth to six children, four of whom were living. December 16th, 1876, she experienced a sensation of chilliness and malaise, but did not think it of sufficient import to inform her physician, whom she had consulted in reference to a sick child that very day. On the evening of the 18th inst., she felt very sick and began to suffer from painful deglutition. Dr. Conrad was called on the following day and diagnosticated diphtheria on both sides of throat,

with moderate fever. On the 20th, I saw her with him. Temperature about 102.5° F., isolated diphtheritic deposits on both tonsils, and a moderate swelling of the cervical lymphatic glands of both sides. 21st: No longer simple gray deposits, but a dense membrane covered the tonsils, extending over the entire uvula and invading a portion of the posterior wall of the pharynx. On the 22d, the membrane was thicker, denser, and had extended forward over one-third of the soft palate and covered the entire visible portion of the posterior wall of the pharynx. That evening slight dyspnoea was experienced by the patient. At eight o'clock on the morning of the 23d, tracheotomy had to be resorted to. It was performed under chloroform, and owing to the displacement of the trachea by the swelling of the thyroid body, it was accompanied by the loss of several ounces of blood. Fifteen minutes after the operation a cylindrical mass about two inches long became loosened and was expectorated; judging from its diameter, it came from one of the bronchi. On this and succeeding days, portions of membrane were cast off. On the 23d, a feather introduced through the canula, by contact with the mucous membrane immediately below the lower end, gave rise to coughing. On the 24th coughing could only be excited by carrying the feather three inches lower down. During that day the dyspnoea, in spite of frequent inhalations of oxygen through the canula, was painful to witness. One-third of a grain of morphia was injected hypodermically at seven o'clock in the evening in order to relieve the sufferings of the patient. She died at ten o'clock. The treatment had consisted in half-grain doses of carbolic acid every half-hour, in the administration of chlorate of potassa and chloride of iron at similar intervals, in disinfectant injections into the nostrils, in half-hourly intervals, of a weak solution of carbolic acid (1 : 50-100) and a spray of a solution of lactic acid (1 : 24) directed through the canula into the trachea and bronchi, every five or ten minutes.

I do not know that sex exerts any predisposing influence over diphtheria, yet of the 600 cases or thereabouts of laryngeal diphtheria in which I either personally performed tracheotomy, or observed the progress of the disease in the practice of others, I found the majority in males and the recoveries in inverse proportion to the number thereof; the mortality being greater among boys. As far as age is concerned, most zymotic diseases are seen most frequently in children. This has been explained by some on the ground that these diseases, particularly scarlatina and whooping-cough, less constantly measles, occur but once in a lifetime, and therefore the number of adults that can still be attacked by those diseases is proportionally small. Typhoid, too, is by no means of rare occurrence, but its invasion is, in my opinion, intimately connected with certain anatomical and physiological conditions. That part of the lymphatic system, Peyer's patches, which plays so important a part in the symptomatology of typhoid, is but little developed in childhood. In addition to this, the act of respiration up to a certain age is performed very superficially, while at the same time the tissue metamorphosis is quite active. In this way the noxious elements are but slowly taken up by the lungs, the rapid metamorphosis of tissue brings about a speedy elimination of the poison, the intestinal symptoms assume no importance, the entire course is a mild one; in fact all the milder the younger the patient. The new-born and infants at the breast rarely take the disease. I saw typhoid in a child of seven months still at the breast. I presented the specimen from a child of sixteen days at the Obstetrical Society of New York, seven years ago, typhoid having occurred soon after birth. It is the only specimen that I have ever seen and is one of the very few that have ever come to light. This subject of typhoid fever I have taken as an example to prove the dependence of a morbid process on certain anatomical and physiological conditions. The same holds true for diphtheria. Children exhibit a greater predisposition toward

the disease than adults, if we except those under ten months. Where the disease has, however, occurred previous to the seventh or eighth month, the greater number of cases has been found under three months. Tigri reports the disease in a child of fourteen days. A child of fifteen days was seen with diphtheritic laryngitis and œsophagitis by Bretonneau, one of seventeen days by Bednar, of eight by Bouchut, one of seven days by Weikert; Parrot mentions several cases, and Sirédey (Thèse, Paris, 1877) reports eighteen cases of diphtheria in the newly born. They occurred in the Hospital Lariboisière in the spring 1877, and were probably infected by the nurses of a neighboring child's asylum. Membranes were found on the soft palate, tonsils, or larynx, and also on both pharynx and larynx. One case occurred where the posterior nares alone were affected. I have met with three cases of diphtheria of the pharynx and larynx in the newly-born myself. One of these became sick on the ninth day after birth and died on the thirteenth day; the other died on the sixteenth day after birth; the third was taken when seven days old, and died on the ninth day. A very young child on whom I performed an operation for complicated hare-lip was reported to me as dying from diphtheria, a few days after, by a young colleague in attendance. It turned out to be muguet, however, and subsided under the appropriate treatment. The predisposition toward diphtheria during childhood* seems to be explainable by several circumstances of the process, whether it develop from within or be derived from external sources. The mucous membrane of the mouth and pharynx in the child is more succulent and softer, and frequently the seat of a congestive and inflammatory process. The nasal cavities are small and frequently affected by catarrhs, the buc-

* W. N. Thursfield (L. Lancet, Aug. 3d, 10th, 17th, 1878) collects 10,000 cases of diphtheria in England, between the years 1855 and 1877. Of these were 90 p. m. under a year, 450 p. m. from 1-5 years, 260 from 6-10, 90 from 11-15, 50 from 16-25, 35 from 26-45, 25 p. m. were 45 years and over.

cal cavity often the seat of catarrh and of stomatitis, and insufficient cleanliness leads here to irritation of the mucous membrane. Any abnormal state of the mucous membrane, with the exception of an atrophic condition and cicatricial changes, affords an excellent abode for diphtheria. The tonsils are proportionally large; in fact we rarely see the tonsils in children completely sheltered by the arches of the palate. On the other hand, the pharynx is anything but spacious, and while the protuberant condition of the tonsils affords a resting-place for the disease, the remaining space is so small that it becomes a source of uneasiness to the well, in many instances, and very much more than that to the child during diphtheritic tumefaction. Furthermore we must take into consideration the large number and size of the lymphatics, which can be more easily injected in the child than in the adult, according to Sappey, and the fact of greater intercommunication amongst the lymphatics and between them and the system. For S. L. Schenck has found that the network of lymphatics in the skin of the newly born, at least, are endowed with stomata, loopholes through which the lymphducts can communicate with the neighborhood, and vice versa (*Mittheil. aus d. Embryol. Instit., I., 1877*). These circumstances, although they may have no influence in calling the disease into existence, yet assist in its development and in adding to the severity of the symptoms.

On the other hand, while the above reasons go to prove that diphtheria attacks children by preference, there is again an anatomical and physiological condition, to wit, the free slightly acid secretion of the mouth, beginning with the third month, that acts as a hindrance to the frequent occurrence of diphtheria after the third month. A poison, or poisonous product of whatever nature can less readily find a hiding-place so long as it can be readily, we might always say must surely be, washed away. An important etiological consideration is the fact of having had the disease previously. We can cite a host of zymotic

diseases the occurrence of which once, serves as a protection against future attacks. Not only can no such security be expected after one attack of diphtheria, but *cæteris paribus*, the disease shows a preference for those who have survived a previous attack. The statement that only the mild cases with but slight elevation of temperature and freedom from severe constitutional symptoms are likely to suffer a relapse is founded on error. True, I have more frequently seen relapses after mild cases—which fortunately are in the majority—but the disease has also recurred where originally high fever and an extensive lymphadenitis proved it to be a severe case. Yet not many can point out a child on whom tracheotomy had twice been performed, as for instance Guersant, whose case has often been quoted, and H. Z. Gill (Abstr. of the Rep. of the Spec. Comm. on Croup of the Ill. State Med. Soc., Chicago, 1878). He operated on March 21st, 1877, removed the tube on the 27th, and the wound was nearly closed on the 28th. On May 11th, the same operation had to be performed for the same disease, with the same favorable result (though the tube was not removed on Sept. 25th, 1878). Quincke also operated on a boy of one and a half years on the 2d of March, 1874, and removed the tube after seven weeks; and for the second time on the 12th of July, 1875, the tube being required two years and a half after (Corresp. Schweizer Aertz., 15th April, 1880).

As there are individuals, so there are families which have a predisposition to diseases, as there are others in whom, notwithstanding ample exposure, infection does not easily take place. Yet in the families in which diphtheria is of frequent occurrence it cannot always be attributed to enlarged tonsils and a tendency to pharyngeal catarrh. My experience has been similar to that of Révilliod, who made this question the subject of a paper read before the Geneva Med. Soc., Dec. 1st, 1875. A certain family with which I am acquainted lost in the course of six or seven years all its younger members with diphtheria of the

pharynx and larynx. The children had been born in intervals of several years. While one child of a few years of age would be affected with diphtheria, there would be another at the breast. After two children had succumbed to the disease at an early age, Dr. Krackowizer performed tracheotomy on the third, who also died. Some years later, I operated on the fourth child, with the same result. I may add that there could be no question of an epidemic or endemic influence, for the cases extended over a number of years, and the family had occupied two different houses in the mean time, in different parts of the city. One child only had never undergone a severe attack of diphtheria, but she was six years older than the one who had been the first victim of the disease.

In diphtheria, as in other diseases, sudden changes in the temperature of the atmosphere, or of the surface of the body, have been looked upon as etiological factors. Yet they have occurred from time immemorial without being accused of causing an invasion of diphtheria, unless when present at the same time as an epidemic. On the whole, however, it is true that, while severe epidemics have spared no climate or land known to us, the majority of cases have occurred in winter and spring, in other words at a time when catarrhal disorders are of most frequent occurrence. Thursfield, while finding no connection between the rate of mortality from diphtheria and the amount of yearly rainfall, points to the variation of mortality in the several quarters of the year. In England, the average mortality from diphtheria in the years 1870-1877 was, in the first quarter, 735; in the second, 578; in the third, 547, and in the fourth, 750. In Lynn, Mass., J. G. Pinkham (Sanitary Condition of Lynn, including a special report on diphtheria, Boston, 1877), found 23 per cent of all the cases to have taken place in October, the smallest number in August. Woizechowski and Aschscharamoff (Mosk. M. Gaz., Nos. 6-8, 1878) counted in 9,858 cases occurring in the year, 5,579 in September, October,

and November. Seitz also finds more cases in October than any other month. In my experience, at New York, the first quarter of the year yielded more cases than any other. But the difference in numbers varies so much with the writers, in different countries and climates, that E. Besnier's attempt at establishing an "invariable season law" for diphtheria (*Un. Méd.*, No. 129, 1878) is liable to appear like rather too much generalization from the experience of one man. Thus looked upon, the influence of season on the invasion and course of diphtheria is but indirect and conditional, and may be perhaps, after all, compared with that exerted by "filth," a term which is lately used to express all sorts and forms of nastiness, from filthy bodies of men, to their clothes, their habits, their food, and the air they breathe, whether polluted by carbonic acid, by excrementitious gases, or exhalations of sewers.

Cases of diphtheria which are traced to exhalations from sewers (or even to filthy habits of life) are very frequent. Yet typhoid is attributed to the same causes. So is dysentery. Can then foul exhalations produce alike diphtheria, typhoid, and dysentery? Do these diseases arise from a common poison? Or is the poison of a treble character, so that a part may give origin to diphtheria, the other part to typhoid, the third to dysentery? In a house in West 22d street, between Eighth and Ninth avenues, in New York, three children and a female help were taken sick, two with dysentery, two with typhoid, in the course of a month. In the same house, in two of the children, diphtheritic sore throats were of frequent occurrence.*

Have we to deal, in such occurrences, with specific influ-

*On the other hand, Thursfield shows that fatal diphtheria is mainly—in England—an attribute of the country, while typhoid fever belongs to the large cities. These, like London, Liverpool, Bristol, have a high mortality of typhoid fever, while Sussex, Shropshire, Oxfordshire, lose a larger percentage of diphtheria. During the years 1870–1877, Liverpool lost at the rate, of a population of 100,000, of typhoid fever 104.3, and of diphtheria 10.3; Sussex, however, of diphtheria 21.1, and of typhoid fever only 31.5.

ences, or only with a lowering of the standard of health, thereby affording other morbid influences an opportunity to exercise their power? These questions are still involved in darkness and constitute problems the solution of which still engages the minds of both individual writers and authorities.

Only recently the results of a careful comparison of a large number of cases of diphtheria reported in Massachusetts were formulated as follows:

1. Diphtheria is contagious, and highly so under certain circumstances. It is also infectious, although not to such a degree as scarlatina, measles, and small-pox.
2. The other circumstances being favorable, a moist soil assists in spreading the disease, be the moisture a natural condition or brought about artificially, and particularly when the substratum is of an impermeable nature.*
3. A positive connection between diphtheria and filth cannot be verified, although the latter but adds to the evil influence of moisture. The contamination of spring-water by human excrements, and of the atmosphere of the bed-chamber by the emanations from sewers, require further study. Several reports point to septic infection by drinking contaminated water, but a final opinion on that point would as yet be premature.
4. Cold and dampness constitute an etiological factor in children, and in individuals with a predisposition toward the disease. Yet the statements concerning wind, temperature, and weather do not allow of any definite conclusions.
5. Other circumstances being equal, natives of Massachusetts and strangers are affected alike.

*J. G. Pinkham reports 614 cases of diphtheria in Lynn, 80 per cent of which took place in valleys of brooks, in the vicinity of marshes, where the soil was damp and without artificial drainage. The immediate humidity of the atmosphere had no influence, however; nor was elevation of any account except in determining the condition of the soil. In all the endangered places the subsoil was an impervious clay.

6. Differences in the course and termination of the disease depend on the idiosyncrasy of the individual or family, on age and on strength.

7. Atmospheric conditions exert an influence which is not yet thoroughly comprehended.

8. The period of incubation, where it was possible to determine it, was about one week.

9. The invasion was sudden and accompanied by marked general disturbances. Death resulted from suffocation or collapse. Convalescence was slow.

10. In adults the disease occurred less frequently, and in a milder form than in children. The disease was seen in babes of five, seven, and nine months.

11. As during the prevalence of epidemics of typhoid and cholera, we encounter mild fevers and diarrheas, so during an epidemic of diphtheria there are always a large number of inflammatory affections of the throat.

For the sake of completeness, I have here given the principal points of the official report, even though some of them are not exactly in place.

I believe that it goes just so far in the estimation of the value of the etiological factors of the disease as our knowledge and experience allow. The assertion that diphtheria is a "filth disease," and the oft-repeated discussions on this point, both publicly and privately, in England and America, cause one to be thankful for the above modest and sensible résumé of our knowledge on the subject, which, on that point, has not progressed much since that report was published, a few years ago.

What is the nature of the diphtheritic poison? Is it of a chemical character? Is it organic?

Already in 1840, Henle expressed his belief in a *contagium animatum*. After morbid processes had already previously been compared with the phenomena of fermentation, Schwann demonstrated the presence of lower organisms in fermentation and putrefaction. The discovery of the cause of the silk-worm disease by Bassi, of the acho-

tion by Schönlein, of the acarus by Simon, of bacteria in malignant pustule by Pollender, Brauell, and above all by Davaine, in relapsing fever by Obermeier, the teachings of Pasteur concerning the conditions under which putrefaction occurs, all tended to explain the various infectious and contagious diseases, by analogy, in the same way, and particularly to stimulate the search for a vegetable organism in diphtheria. Buhl was the first to discover schizomycetæ in diphtheritic membrane, but expressed no opinion as to the part they played in the process. Hüter found them in the gray diphtheritic covering of wounds, in the surrounding, apparently healthy tissues, and in the blood. Hüter and Tomasi found them in the diphtheritic membranes of the pharynx and larynx, inoculated them on the mucous membranes of animals, and described them as small, round or oval, dark-colored, active little bodies. The latter observers look upon these organisms as a part of the infectious element. Oertel found them in diphtheritic membrane and in inflamed mucous membranes in the lymphatic vessels, lymphatic glands, kidneys, and other organs; he considers them at the bottom of the diphtheritic process and constituting the contagious element. Nassiloff, too, after inoculations in the cornea resulted in an enormous multiplication of the microscopic organisms, and their appearance with pus-cells in the lacteals, and in the lymphatics of the palate, and even in the bones and cartilages, asserts that the development of organisms is the primary step in the diphtheritic process. Eberth made successful inoculations in living tissues; the micro-organisms, introduced into the cornea, proliferated actively and caused an inflammation of irritative character in the surrounding tissue. He asserts, with the positiveness of an evangelist, that diphtheria cannot occur without bacteria. Klebs inoculated the micrococci in pigeons and dogs and demonstrated the presence thereof in the blood of the animals after death. Orth found them in the pleura, lungs, kidneys, and urinary bladder. But what their action is, whether they are

directly pernicious, or deprive the body of certain elements (as of oxygen in malignant pustule, according to Bollinger), or injure mechanically, by acting on the coats of the blood-vessels either directly or by means of altering the blood, and thus ruling out of existence territories of blood-vessels, is a question upon which the principal advocates of the parasitic theory have not yet agreed. Even Oertel acknowledges the impossibility of explaining the manner in which the bacteria act (Ziemssen, *Handbuch*, II., 1, p. 581, 2d edit.). This much is positive at any rate, that no one has yet proven that the vegetable organisms alone, and not other, free or fixed, parts of the diphtheritic membrane, are the vehicles of the infecting elements (Steudener); and even now the question has not been decided whether the bacteria met with in diphtheria constitute the cause of the disease; or are a part of the process, or co-effects of the poisonous action; whether they are the carriers of the poison, or entirely indifferent entities.

The most important observations made by those who deny a direct etiological connection between micro-organisms and septic diseases in general, and diphtheria in particular, are those of Hiller, and Billroth. The latter has proven the morphological identity of the various kinds of bacteria, although it cannot be denied that the apparent similarity may mask a yet unknown difference. Hiller calls attention to the fact that large numbers of micrococci have been found in the cadaver where death has not been the result of septic disease, and also that septic infection is not always severest where the bacteria most abound, but where an extensive chemical decomposition or a mass of putrefying tissue is found. This would indicate that the septic process is rather dependent on chemical decomposition than on the presence of bacteria.

Panum, Bergmann, and Schmiedeberg have isolated poisons that contained no bacteria. Rawitsch and many others prove that septic infection is not dependent on the existence of bacteria. Davaine has shown that an infi-

nitely small amount of a chemical poison, free from bacteria, can kill quickly. Panum, Billroth, C. O. Weber, and Hemmer emphasized the chemical nature of the poison. Hiller, by injecting isolated bacteria in large numbers into the subcutaneous cellular tissue of dogs and rabbits, produced a slight local swelling, but neither abscesses nor fever. Even injected into his own subcutaneous cellular tissue, it produced but a slight œdema; in subcutaneous wounds which would not have suppurated otherwise, only œdema was produced. Experiments with the true bacteria of putrefaction led to a similar result. Injected into hens' eggs, the latter did not become rotten. There must be, therefore, elements of putrefaction which are independent of bacteria, and Pasteur acknowledges the truth of this remark. So much is true, that bacteria do not receive their albumen in the form in which it exists in the tissues, but it must be first dissolved by the putrefactive process. Therefore, in diphtheria the gangrenous process cannot or need not depend on a direct "devouring" of the tissue. Hiller, from observing that, in accidental diseases of wounds, bacteria are a frequent and probably constant accompaniment, and that their most active proliferation ensues after death, believes that they fill themselves with the septic poison of the decomposed tissues, and then transport it further, in this way exerting a local as well as a general action; and also that it may even be in their power to reproduce the poison, but that they do not carry an independent danger of their own, or essential to them, and, as parasites, may be considered harmless. If any organic substance capable of undergoing putrefaction be separated from the original tissue, it will certainly float in the air in the form of an atom of dust, yet an infection need not always result. But these elements of putrefaction are probably not bacteria, but some other molecular body accompanied or not by bacteria. Besides, we cannot yet assert with conviction that the agents of infectious disorders are actually solid, still more, organic bodies. As they

may just as well be fluid or even gaseous, the nature of infection is still in the dark.

Billroth, too, holds that plants cannot decompose organic compounds, and can only digest gases already formed; that bacteria only serve to excite an inflammation when they themselves are infected, and if introduced into the cornea they assume the characteristic form of the fungus and may then prove innocuous; that cocco-bacteria, a foul secretion, and fever are not always found together. "The secretions from the earlier stage of inflammation have a more powerful phlogogenous action than those of a later period, and the action of the micrococcus does not depend on its mechanical influence on the tissues, or on its presence there in a living form, but on whether it is the means of conveying irritating elements into the tissues or not." (Arch. f. Klin. Chir., XX., 414.) Ehrlich, also, in his experiments on erysipelatous skin, found that the commencing inflammation was accompanied by the formation of a large number of micrococci, which *gradually disappeared as the process progressed*. In addition, when fluids containing cocco-bacteria were injected into the skin, micrococci, and not scattered or closely clustered bacteria, came into view. True, the latter were seen in small blood-vessels, but a cellular infiltration was rarely observed to surround their abode. Fibrinous thrombi were not by far present on every occasion in the smaller blood-vessels, and therefore had probably not formed during life, or if so, only in the last moments. That cocco-bacteria are transferable from one part to another is self-evident. Their minuteness, and the fact that small emboli can traverse the pulmonary circulation and be deposited in remote organs, would indicate and in fact presuppose their appearance in the lymphatic channels and glands. Their circulation in the blood-vessels would be freer than in the lymphatic system, where the glands act as impediments and filters. But if we assume that the transportation of bacteria goes on in the same manner as that of

emboli, we should find the former at the place where the arteries become capillaries, whereas they are found instead in the small veins. Hence this fact would speak rather for a rapid formation after death than for a process occurring during life.

The presence of cocco-bacteria in the blood during life has not once been proven, not even in pyæmia or septicæmia. Yet their being swept into the lungs with the atmospheric air is indisputable. It would, therefore, seem as though living blood had a greater tendency to destroy bacteria than to allow itself to be decomposed by them. Not only, however, would it seem so, but P. Grawitz (*Virch. Arch.*, vol. 70, p. 546) proves that sporules do not grow in the (tissue and) blood, but that they are in part dissolved, in part eliminated through the kidneys, and that this result is accomplished through the combination of the following four factors, viz., the elasticity of the blood, its constant motion, the absence of oxygen in sufficient quantity in the circulating blood, and the presence of living animal cells. All of these factors appear to be of great importance. Thus it is that, where the constant motion of the blood and the animal living cells are not present (as in the anterior chamber of the eye, or in the humor vitreous), a rapid proliferation and accumulation of bacteria can take place. They are also known to increase rapidly, and emigrate into the liver, when deposited in the abdominal cavity.

The destruction of bacteria going on in the circulating blood, into which they may have penetrated, accounts for some microscopical facts in connection with (actually or apparently morbid) blood. Their remnants are probably the pale and dark particles which are discovered in the blood, alongside the red and white blood-corpuscles. They could not be identified as micrococci, while in the tissue they are more recognizable. In autopsies, they have been found in the urinary tubules, pressing forward and piercing the walls, not occupying a nidus of inflamma-

tion, however, and probably, are even here a post-mortem phenomenon. A direct necrosis or inflammation, by the inoculation of diphtheritic elements, can only be produced in the cornea, as was shown by Recklinghausen, and particularly Eberth. Besides, there is nothing characteristic in the cocco-bacteria of diphtheria, with the exception, perhaps, of their browner color, to justify their being looked upon as a distinct variety, certainly not as another species. It is more likely that a difference of action is not so much to be sought for in a different parasite, as in the peculiarity of the corneal tissue. When fluid containing cocco-bacteria was injected into the eye of a rabbit, in twenty-four hours the eye was destroyed. If injected into the eye of a dog or guinea-pig, only a slight inflammation resulted (Billroth and Ehrlich). If these experiments were continued on a larger scale, we might eventually, by analogy, infer and even prove, that the immunity against certain diseases enjoyed by some animals is owing to peculiarities in the very structure of their own tissues.* In a similar manner I shall prove hereafter that even peculiarities and variations in the tissue and epithelium of the human body give rise to different shades and variable clinical symptoms in the diphtheritic processes.

The views of Curtis, Satterthwaite, and Charlton Bastian fully agree with those of the above observers. Bastian, from the occurrence of fermentation and putrefaction without the presence of bacteria (since the analogy between contagion and putrefaction must be maintained), asserts the innocuousness of a multitude of bacteria in the blood, in all parts of the body and in open wounds. He testifies to the inverse proportion between the virulence of some septic fluids and the number of bacteria contained therein, and to the impossibility of depriving a septic fluid of its active properties by boiling and the addition of alcohol. He is rather inclined to look upon bacteria as an effect of the disease than as a cause.

* Compare p. 25.

Curtis's and Satterthwaite's careful and judicious experiments led to the following conclusions (Edward Curtis and Thomas E. Satterthwaite, Report on Investigations into the Pathology of Diphtheria):

I. Inoculation of diphtheritic membrane into the muscular tissue of the rabbit produces severe local lesions, and even constitutional disturbance and death. But these effects differ so in their pathology and clinical history from diphtheria in the human subject that there is no warrant for defining them as diphtheria, or for applying conclusions drawn from observation of this inoculation disease in the rabbit to the case of diphtheria in man.

II. Effects exactly similar to the foregoing, and of equal severity, can moreover be produced by inoculation of a material not only non-diphtheritic, but non-infectious to the human subject, under conditions where diphtheritic membrane is infectious, *i. e.*, when brought into contact with the mucous membrane of the mouth and throat. The material referred to is the pulpy scraping of the upper surface of the healthy human tongue.

III. Effects generally similar to the foregoing, though not of equal intensity, can furthermore be produced by inoculation of a putrescent matter which is not even of immediate animal origin, namely, Cohn's fluid, allowed to spontaneously decompose (an aqueous solution of ammoniac tartrate, potassic and calcic phosphates and magnesian sulphate).

IV. The foregoing inoculation effects are not due to simple mechanical irritation, for inoculation of sand produces no effect whatsoever.

V. Thorough filtration of a proven virulent aqueous infusion of diphtheritic membrane, or of putrid Cohn's fluid, removes the infectious property of the same. Hence in such diphtheritic infusion the poisonous quality probably inheres in some *particulate* thing, from which it is not separable by the action of cold water.

VI. Thorough trituration of proven virulent diphthe-

ritic membrane and tongue-scrapings, with a high percentage of salicylic acid, fails not only to remove, but even markedly to modify, the intensity of the infectious quality of those substances. Hence, since salicylic acid, in even a minute percentage, is capable of permanently suspending the vital activity of bacteria, the inference is that the infectious quality of diphtheritic membrane upon the system of the rabbit is not correlated to the vital activity of the bacteria present in such membrane.

VII. If, as is not improbable, the noxious principle in diphtheritic membrane which produces in rabbits the effects described, be the same with, or even analogous to, the principle which produces diphtheria in man by direct infection, then the conclusion of VI. will apply to the infectious quality of such membrane, in its relation to the reproduction of diphtheria in the human subject. If this be the case, it follows as an important practical corollary that *there is no theoretical ground for assuming that preventing the bacteria of a diphtheritic patch from making their way through the underlying mucous membrane will, per se, prevent general diphtheritic infection of the system.*

VIII. There is no relation between inoculable virulence of a diphtheritic membrane and the period, within three days, that has elapsed between the detachment of the membrane and the inoculation with the same, nor between inoculable virulence and gross amount of bacteria present in the membrane.

IX. There is a rough relation between inoculable virulence of a diphtheritic membrane and the severity of the original case of diphtheria, so far as this can be estimated by the termination of the case in death or recovery.

But, the investigators add, in the true spirit of men who are looking for facts and not for the upholding of a preconceived theory, that "it must be distinctly understood that these nine propositions are not put forth as proven, but merely as the results of our experiments and observations, so far as the latter go, stated in abstract form. Before the

propositions can be considered proven as truths, a large number of corroborative experiments will have to be made."

A similar result was obtained by Burdon Sanderson. The clay filter undoubtedly absorbs the granular contents of the fluid. These particles are either soluble or in a state of suspension in the form of minute granules, in the fluid which is contaminated by their presence, and although it may seem clear to the naked eye, yet will be found, when examined microscopically, to contain minute granules. Septic fluid containing such granules was exposed to the action of alcohol and boiling, both of which are fatal to bacteria. The fluid remained septic notwithstanding, even although, while the circumstances were favorable, no bacteria developed therein. Furthermore, no bacteria could be propagated from the granular bodies.

The experiments of Weissgerber and Perls likewise afford no great encouragement to the partisans of the parasite theory. They studied the influence, on micrococci, of a narrowed calibre of veins, and for the present still left to further discussion the question, whether the clusters of micrococci found in inflamed tissues, as in erysipelas (Lukomsky), and in small-pox (Weigert), are the result of the presence of the exudation which would have the effect of retarding circulation. However, they seem to incline to the opinion that, when micrococci are found in the glomeruli of the kidney, their presence is to be explained by embolism, and when observed in the capillaries and veins, by stagnation. But they acknowledge that there are cases in which micrococci are found in the glomeruli from stagnation also. Many have inclined to the view of an accumulation of bacteria (and then only a proliferation) in embolism, as Lücke asserts in connection with infectious periostitis (*Deutsche Z. f. Chir.*, 1874, vol. 4). But experiments in no wise substantiate this, for the above-mentioned observers having, in a number of animals, narrowed the calibre of the renal vein, and either during,

immediately before, or immediately succeeding the operation injected into the blood in some cases aniline-blue in a state of finest dilution, in others isolated bacteria taken from a putrefying fluid, could not find the injected substances to any large amount in the corresponding kidney, although their presence could be detected in the tissues, and in considerable amount in the other kidney, the circulation of which had been undisturbed. The above results ought to throw a damper on the hopes of those who infer from the presence of micrococci in the kidneys that they are the cause of desquamative nephritis, as was but recently the case with Reimer. Least of all can we pardon an author for declaring a somewhat obscure case of renal disease to be diphtheritic only because he happens to find micrococci in the kidneys; while the whole question is still *sub judice*.

After all the foregoing, when we compare all the evidence adduced for and against the etiological signification of bacteria as originators of infection, we can come to no affirmative conclusion. That the lowest organisms might be important, etiologically, in the infectious diseases was, from the start, a plausible assumption. It has not been possible, however, to find special organisms for different diseases—with the exception perhaps of certain stages of malignant pustule and relapsing fever. At all events, their manner of action is hypothetical; to assert that infection is analogous to the process of putrefaction is equivalent to leaving both putrefaction and infection unexplained. I do not think that even to-day we are justified to go beyond this statement of Panum's concerning our present knowledge on the subject: "It is a matter of rejoicing that physicians in general have come to the conclusion that certain microscopic organisms, be they considered vegetable or animal, and designated as bacteria, fungi, monads, micrococci, or vibriones, do not exist merely in the minds of theorists as causes of disease, but are in reality enemies that must be combated with all the known, efficient weap-

ons in our possession. But while thus rejoicing, it must be borne in mind that we have but a feeble insight into the relation between these organisms and diseases, and in order to effect that much desired advance in scientific knowledge—a matter of considerable importance in the practice of medicine—it is necessary, not only to grasp at isolated data, but carefully and deliberately to observe and study all the facts before us; and even to devote some attention to those which would tend to prove that there are bacteria and fungi which, under certain circumstances, are perfectly harmless, and that even some of the malignant ones among them do not commit all those outrages with which they are charged, directly and personally.”

To what extent this is true is perhaps best illustrated by the results of A. Hiller's investigations, published in his late work on Putrefaction (Berlin, 1879). He cannot be accused of experimenting hastily or writing too early. Thus it has not happened to him to be compelled to change opinions too often. In my contributions to the pathology, etc., of diphtheria (Amer. Jour. Obst., Feb., 1875), I referred to his position in the bacteria question; and I am glad to be able to say that in his late large work he can afford to state, not as his opinion, but as the result of his studies, that the “septic or putrid” poison contained in putrefying materials is a chemical substance formed by the process of putrefaction; that its efficacy does not depend at all on the life or presence of organisms; that the symptoms of septic and putrid infection are observed without the co-operation of bacteria or micrococci; that bacteria cannot be, *per se*, the poisonous principle in putrefying substances, but that this must be a material adhering to (possibly even produced by) the organisms, which can be separated from them by a washing process; that there are numerous cases in which the multiplication of micrococci in the living body was uninjurious, and but the result of the disease; and that any progress of our knowledge concerning the whole subject will depend on renewed investigations of

the metamorphosis of the minute organisms, and of their share in the chemistry of the putrefaction process.

In regard to diphtheria in particular, the parasitists have always proceeded with so much enthusiasm that many promises which were made could not be fulfilled, and many proofs which were offered could not be presented. Klebs does not prove, he begs, his case when he insists upon it that the diagnosis of diphtheria depends on the presence or absence of the microsporon diphtheriticum. A case is not settled by the non-acceptance of a diagnosis for no other reason but the absence of a parasite, the presence or accumulation of which is not expected or claimed as pathognomonic by hosts of others; nor has the promise infallibly to produce the disease on man and animals by inoculation, to reproduce the disease by "synthesis," been made good to the extent promised; nor is the large number of journal essays and contributions apt to sweep away the critical judgment of the profession. Our experience has been that the very latest microscopical investigations of the most prolific writers always claimed to be unimpeachable revelations. One of the most energetic parasitists, L. Letzerich, according to whose dicta the specific parasites of diphtheria, of whooping-cough, of typhoid fever could be diagnosticated under the microscope as though they had been labelled, admits now (*Arch. f. experiment. Pathol. u. Pharmacol.*, vol. XII., 1880) the great difficulty of discriminating the specific schizomycetæ of diphtheria, infectious croupous pneumonia, epidemic influenza, and typhoid fever in their accumulations in the human and animal body. While last year there were no difficulties whatsoever in their ready diagnosis, this year the morphological differences are better appreciated by an artificial propagation and proliferation in the blood taken from the sick, I cannot but feel that the very progressive part of the profession is also the conservative one, and will not readily admit that a few microscopical investigations, instituted with no method or with insuffi-

cient methods, or with good methods which will be improved upon to-morrow, are able to revolutionize, at one stroke, modern pathology. Progress *is* slow, no matter how rapid.

There are schizomycetæ, more or less, in most cases of diphtheria and other infectious diseases. But it is not proven, but only claimed that they are essential. Even when micrococci, by immense accumulation in certain localities, result in a local necrosis of the tissue, it may be claimed, but it is not proven at all, that this effect of the parasites is the essence of diphtheria. We know that in a certain period of relapsing fever the spirochæte discovered by Obermeier is always met with, but we also know that the theory of the disease itself has not yet been materially advanced by the discovery, be it ever so interesting and, let us hope, important. There is, without any doubt, in every anthrax the bacterium anthracis Cohn. Still it is an undeniable fact that the blood from those sick with anthrax transferred to the healthy animal destroys life very rapidly, before a considerable development of bacteria can take place; and also that the poisonous nature of the blood increases in virulence beyond any proportion to the increase of the parasites. That circumstance militates rather for a chemical than a parasitic poison.

Meanwhile, however, the parasitists have accomplished a victory. For the public mind and the judge's bench even is infected with bacteric faith. Bacteria are recognized by jurisprudence. The Berl. klin. Woch. of August 2d, 1880, refers to a forensic case, in which an unlicensed practitioner was found guilty of "having permitted, by his therapeutical procedures, the bacteria to enter the body of a patient, and caused her death thereby."

SUMMARY.

Diphtheria is pre-eminently a disease of childhood. It is not frequent amongst adults, very rare in old age.

It is not frequent in the first year. Still there are, for

physiological reasons, more cases before the third month than between the third and seventh or eighth.

The sexes are liable to be taken in about equal proportion. Laryngeal diphtheria is more frequent in boys. Recoveries from it in girls.

Diphtheria is apt to recur in those who once had it. Even membranous croup has been observed twice in the same patients.

Some individuals, and even families, have a certain degree either of immunity or predisposition.

Exposure and "colds" may act, but as proximate causes only. Most cases take place in the winter months in our climate, but there is no "invariable season law."

"Filth" contributes to the generation of diphtheria, as it does to dysentery and typhoid fever.

The question of a live origin of contagious disease in general was raised by Henle in 1840, also by Sir H. Holland, and Eisenmann.

Some pathologists find the morbid source of diphtheria in bacteria. "No bacteria, no diphtheria." This is not truer than that fermentation or putrefaction depend on bacteria only.

The presence of bacteria in the diphtheritic blood has not been proven. There is no theoretical ground for assuming that preventing the bacteria of a diphtheritic patch from making their way through the underlying mucous membrane will, *per se*, prevent general diphtheritic infection of the system. On the contrary, the septic and putrid poison is claimed by A. Hiller as distinctly chemical. Of the same nature, viz., chemical, is very probably the poison of those of the infectious and contagious diseases in which the presence of a characteristic parasite is a recognized fact, as anthrax and relapsing fever.

CHAPTER III.

THE MANNER OF INFECTION.

The view that diphtheria is a general infectious disease of the blood, rather than the result of a local infection, its upholders attempt to prove by a host of reasons which may be summed up as follows :

Diphtheria occurs epidemically. This rule is not in collision with the fact that there are sporadic cases likewise, for the same holds good for measles, scarlatina, and small-pox. The principal objection to it, however, lies in the fact that diphtheria has not a typical course, and that so far from one attack of the disease offering a security against its recurrence in the future, the disease has a tendency to revisit those who have undergone a previous attack.

Epidemics of this disease, as of others, differ in duration and severity.

There is a period of incubation which, it is true, varies from two days to two weeks.

Diphtheria is communicable by contagion and inoculation. Yet this can be said of every local contagious skin-disease, and the diphtheritic poison behaves in the same manner as the *acarus scabiei*.

Constitutional symptoms, as pain in the limbs, lassitude, chilliness, fever, and convulsions may be present for hours and days before the appearance of the local symptoms; and symptoms of general poisoning and adynamia are frequent.

Albuminuria is a more frequent and earlier occurrence than the height of fever, if present at all, would explain.

Furthermore, the frequent occurrence of paralysis as a sequela of the disease would speak for an attack upon the general system from the very outset.

The study of the manner in which diphtheria effects an entry into the body, whether by a primary poisoning of the blood or by local infection, would perhaps afford the most ready solution of the entire question. But one case does not explain all, and diversity of opinion or observations ever so correct, would only prove the diversity of natural occurrences. So much is positive, that there are certainly some cases in which a local affection is the first symptom of the disease. Mercado's old case of diphtheria, engendered by the biting of a finger, has been alluded to in Chapter I. I know of one case in which the vagina became first affected and later the pharynx. Bayles saw denuded portions of skin assume a membranous character, and general diphtheria develop afterwards. Fresh wounds become diphtheritic and the general disease arises from this source. Even paralysis will follow. I had a death from diphtheria when a long incision into a phlegmon of the thigh had become diphtheritic. A little girl, who had a considerable amount of discharge from a catarrhal vagina, and sore thighs in consequence, exhibited first, during the prevailing epidemic of 1877, membranes on the denuded cutis, and afterwards general diphtheria. The boy, whose case I shall relate later as one of diphtheria of the pharynx, had general symptoms of diphtheria, fever, and pharyngeal affection. H. Brehm reports the case of a woman on whom he performed colotomy. The wound became thoroughly diphtheritic and gangrenous, but the pharynx and respiratory organs remained intact. A few days after, her daughter, who attended her in her sickness, was infected. In her, the pharynx was the seat of disorder. Besides, the tonsils are very frequently coated with a membrane without any general symptoms in the beginning, fever and general illness occurring only later on. Now all of these facts

tend to show that *there are cases in which the origin of the disease is purely local.*

It must not be forgotten that during the prevalence of an epidemic every one is more or less under its influence, and but little is wanting to call forth the disease. Some years ago, a well-known physician, with whom I was intimately acquainted, died from facial erysipelas and meningitis, which had originated in a slight abrasion of the upper lip. During an epidemic of typhoid we daily see persons with fever, headache, and lassitude. Diarrheas are frequent during an epidemic of cholera. An epidemic of diphtheria is accompanied by a great number of cases of pharyngitis. When I, in the year 1860 (*Amer. Med. Times*, Aug.), reported two hundred cases of *bona fide* diphtheria, I at the same time observed one hundred and eighty-five cases of non-membranous inflammations of the throat. Therefore, contrary to the view of a local origin of diphtheria it may be claimed that the individual taking the disease was already saturated with the poison, and the local membrane represented perhaps nothing but a symptom, or at the utmost the *causa proxima*. Accordingly, then, there are undoubtedly cases in which the pharyngeal membrane is the first cause and symptom of the final affection, and others in which the poisoning of the blood through inhalation is the first step in the development of the disease, amongst the symptoms of which the pharyngeal membrane counts as one.

The view that many cases of diphtheria are constitutional affections is supported by the fact that the first complaints of the patient relate to the general condition. The symptoms will be described later. Sometimes the patients are ignorant of any throat trouble when they consult a physician. On the other hand, it is true that the throat and respiratory passages in general are usually and, in fact, almost exclusively the seat of the visible affection, and this would seem to indicate that the infec-

tious elements while being inhaled are deposited on some of these parts, and are there developed and set in action; in other words, favoring the view of a local action. This facilitates the possibility of simultaneous affections of both the throat and the blood in the lungs, in either equal or variable proportions. We are easily led to defend at least a partial admission of the poison by the respiratory act, when we reflect that the membranes which are swallowed are rendered innocuous by the action of the gastric fluids, and, therefore, the alimentary canal, from the œsophagus downwards, cannot be made responsible for the admission of the poison. Thus it is that the general symptoms—as fever, lassitude, etc.—precede the local phenomena in very many cases; while there are exceptional cases in which the membrane appeared first, and the fever later. This is especially the case when the tonsils are very large and occupy a prominent position in the throat. The difference in the invasion of diphtheria can be clearly illustrated by the following cases: Two boys, in a certain family, became sick at the same time; one, whose tonsils were small and barely visible, suffered from fever and headache, but the membrane could hardly be distinguished. In the other, whose tonsils were large and very prominent, they exhibited an extensive membranous layer, but he was free from fever. On the second day, the former of the boys had a distinct membrane on his tonsils; the latter was feverish. Thus individual cases must be compared with reference to the elevation of temperature, the extent of the membrane, and the longer or shorter duration of the disease, and their significance and probable origin estimated.

Those cases which begin with high fever and moderate or even no local symptoms must be looked upon as constitutional diseases, brought about in this wise: The vast ocean of blood in the lungs, with a surface of two thousand square feet, which contains a fifth part of the entire mass of the blood, is separated from the vitiated atmo-

spheric air only by the thin epithelial layer (which is even absent at times) of the alveoli of the lungs, by a delicate basement membrane and by a fine capillary network. In this connection it is immaterial whether diphtheria be ascribed to a parasitic or a chemical infection. The latter, when we remember the destructive effect the alkaline blood has on bacteria, would best explain, by a saturation of the blood, either direct or catalytic, the symptoms of the disease. In cases accompanied by a high fever and a slight formation of membrane, no other explanation would be satisfactory. Of course, there may be various degrees of invasion.

Should a person, in the course of several hours or a day, be taken with high fever and a moderate membrane formation, these symptoms subsiding in one or two days, leaving the patient weak and exhausted, but fully restored to health at the end of a week, we would be justified in assuming—*cæteris paribus*—that there was a rapid absorption of a large amount of poison, and an equally rapid elimination thereof. Thus, my colleague, Dr. Conrad, found the temperature in the axilla of a patient to be 107° F., after eighteen hours, 102°, and in a few days, 99°. The same experience I have often had. They are, moreover, the same cases, in which the second or third day of the disease furnishes albuminuria, with rapid elimination and speedy recovery. When, however, the process is slow in developing, accompanied by moderate fever, and the course is indolent, we have reason to infer that moderate amounts of the poison are being continually taken into the system, and making their influence felt to a moderate degree, but for a longer period. Such are the cases which, without any violent symptoms, are accompanied by frequent local relapses, or run, when the absorption is constant as well as copious, a septic course, or terminate in paralysis.

These are my views on the question of a general or a local cause of diphtheria. There are cases in which a

local infection of the skin, or of a wound, may be one of the causes, or the only cause, of the disease, and there are cases in which the poison, in passing through the pharynx, gives rise to local phenomena before the system at large gives evidence of infection. But as a general thing diphtheria must be looked upon as a constitutional disease, giving rise to local phenomena, in the same way as scarlatina does on the skin, on the mucous membrane of the alimentary canal, and in the uriniferous tubules; measles on the skin and respiratory mucous membrane, or typhoid in the lymph follicles and on the mucous membrane of the intestine; or in other words, the diphtheritic poison may enter the system locally through a sore or wounded integument, or through the lungs.

SUMMARY.

The entrance of the diphtheritic poison into the system is not the same in all cases.

There are cases in which the origin of the disease is decidedly local.

There are others in which the poisoning of the blood through inhalation is the first step in the development of the disease.

In many cases, both a sore integument and the lungs are the inlets of the poison simultaneously.

It is probable that the configuration of the vestibules of the respiratory apparatus, and the amount of active poison, and the duration of the exposure to it, modify the intensity of the symptoms and the course of the disease.

CHAPTER IV.

CONTAGION AND INCUBATION.

That the contagiousness of diphtheria should still be doubted is hardly possible, and still the public act as if it did not exist. One of the latest facts is that communicated by Trammer to the annual meeting of the Illinois State Medical Society, on May 18th, 1880 (Med. Rec., June 12th). In one school district, with 59 pupils, an epidemic was started (no cases having previously existed) by two boys who visited a neighboring community where there were cases of the disease. In a few days, both boys had symptoms of cold, received some domestic treatment for their little fever and sore throat, and soon returned to school, where other pupils complained of the offensive odor of their breath. Soon other cases appeared, and the number of persons attacked was 58, with 17 deaths.*

That diphtheria is contagious is beyond doubt.† The contagious element is directly communicated by the patient; it clings to solid and semi-solid bodies, and in this way is transmitted even after a long time. There is hardly any disease which can cling as tenaciously to dwellings and furniture; it can be transported by the air, though probably not to a great distance, and hence in houses artificially heated, while the windows and doors are

* There were 17 cases of nasal diphtheria, with 15 deaths. The treatment is reported as consisting of injections of a 3 to 4 per cent carbolic acid solution, and salicylic acid, tannin locally, and bromine, according to Wm. H. Thompson.

† Though there be a man in Vienna who not only treats diphtheria as non-contagious in placing diphtheria patients into general hospital wards, but also writes pamphlets in that sense—or nonsense.

mostly closed, rises from the lower to the upper stories, and it is for this reason advisable to keep the sick on the top floor. It is certainly transmitted by spoons, glasses, handkerchiefs, and towels used by the patient. The contagious character increases directly in proportion to the neglect of proper ventilation. A. Carpenter (*Brit. Med. Jour.*, Jan. 4th, 1879), looks for a predisposing element in the development of diphtheria even in the accumulation, in dwellings and school-rooms, of carbonic acid gas. Fresh air, plain diet, sulphurous acid, and creasote are therefore the preventives. Workmen in gas factories, according to him, do not suffer from diphtheria. That it is spread by the feces is not clearly established in my mind. I can give personally no examples of its being spread by visitors or by the attending physician; this is said to have occurred, however. The character of the disease communicated, and the local manifestation, do not depend on that of the original sufferer; thus mild cases may produce severe ones, and vice versa; and convalescents can convey the disease in its full force. Naturally the softer character of the tissues in children renders them more susceptible to infection, and the activity of their lymphatic system liable to severe forms of the disease.

Many tragic cases are recorded in literature, of infection by direct contact from pharynx to pharynx, or from the opening in the trachea to the mouth of the surgeon, and one of the saddest cases, perhaps, is that of the much lamented Carl Otto Weber. I became affected with diphtheritic pharyngitis followed by a tedious catarrh, consequent upon sucking the wound, during the performance of tracheotomy, in an eight-year-old child, who died thirteen days later of gangrene of the trachea and anterior portion of the neck, and general septic poisoning. The infection in my case resulted in the outbreak two days after sucking out the wound. Oertel has seen diphtheria of the pharynx which was communicated by the act of kissing,

and developed in two days. A series of cases in which the surgeon was infected by membranes expelled during the performance of tracheotomy, and where the period of incubation was from two to three days, have been reported, particularly by French physicians. Dr. Symington, House Physician at Bellevue Hospital, New York, suffered in 1876 from a diphtheria of the pharynx followed by paralysis of the soft palate and of the extremities, which lasted for months, after exposing himself to direct infection from a tracheotomized windpipe. In his case, too, a period of two days elapsed between infection and the appearance of the disease.

In regard to the length of the incubation periods, there can be no better authenticated facts than those contained in a report of Dr. Elisha Harris to the National Board of Health, an abstract of which is found in No. 1, National Board of Health Bulletin, June 28th, 1879. The report says that, in the fourth school district of the township of Newark (Northern Vermont), amidst the steep hills, where reside a quiet people in comfortable dwellings, the summer term of school opened on the 12th of May. Among the twenty-two little children who assembled in the school-room in the glen were two who had suffered from a mild attack of diphtheria in April, and one of them was, at the time school opened, suffering badly from what appeared to have been a relapse in the form of diphtheritic ophthalmia. Besides, it is proved that these recently sick pupils had not been well cleansed, one of them having on an unwashed garment that she had worn in all her sickness three weeks previously.

At the end of the third day of school, several of the children were complaining of sore throat, headache, and dizziness, and on the fourth day and evening so many were sick in the same way that the teacher and officers announced the school temporarily closed. By the end of the sixth day from school opening, sixteen of the twenty-two previously healthy children became seriously sick

with symptoms of malignant diphtheria, and some were already dying. The teacher and six of the pupils were not attacked, nor have they since suffered from the disease.

The Würt. Med. Corresp. Bl., 1878, No. 2, reports the case of a surgeon who, while attending a diphtheritic child, had some secretion thrown into his face. Twelve hours after, his right eye was inflamed and painful. After thirty-six hours, the conjunctiva bulbi exhibited a diffuse redness, and secreted a turbid fluid mixed with shreds. The cornea was disfigured by several ulcerations. Photophobia and much headache. Eserine 1:100 was used six to eight times daily; thirty-six hours after, while the right eye was improving, the left was affected similarly but less seriously. The same treatment was continued, and recovery took place after some weeks, nothing remaining of the affection except a few yellowish spots and some photophobia.

It would then appear that, in the direct communication of the disease to healthy or nearly healthy mucous membranes—as healthy as the prevailing epidemic will allow—the period of incubation is two days. In only a small number of cases, the disease has an apparently shorter period of incubation than this; as when tonsillotomy or a similar operation is undertaken during the prevalence of an epidemic. One may rest assured that any operation on the tonsils, while an epidemic of diphtheria is at its height, will be followed in twenty-four hours by diphtheritic deposits on the operated part. To what extent we are justified in considering this a *bona-fide* incubation of the disease in a previously healthy body is, of course, another question. It seems to me that these cases positively prove that the operation is only the *causa proxima* of a diphtheritic affection, and that we have rightly taken it for granted that during an epidemic every individual is more or less under its influence and affected by it, so that it needs but a wound or an accidental abrasion of the surface of the mucous membrane to call the disease into

action. In a similar way fresh wounds, or morbid conditions of the mouth, may call forth the disease. The ruptured vesicles of a follicular stomatitis are liable to serve as resting places for diphtheritic membranes, and thus I have seen the complication of a follicular stomatitis with oral diphtheria; and any lacerations of the vagina during labor may become diphtheritic within twenty-four hours. If now, on the one hand, incubation depends on the condition of the affected surface, it is probable, on the other hand, that the intensity of the poison at the time, plays an important part in determining the period that is to elapse between infection and the invasion of the disease. It is so very difficult to fix the period of incubation in every case—that it should differ in different cases is certain—for the reason that in many the objective symptoms of diphtheria are very unimportant, and the physician's attention is only called to them after a number of days. Moreover, the nature of the infection is still little understood, and the question of a local or a general beginning of many individual cases of the disease still remains a mooted point. The fact that a number of constitutional symptoms may precede the outbreak of the local infection would at once indicate that a certain class of cases is *most assuredly dependent on a general infection, and cannot be traced to a local influence*. If this be true, it becomes a difficult matter to determine the period of incubation. During an epidemic there are many possible modes of infection, and diphtheria in so far resembles other infectious diseases that it does not require a real bodily contact to take or give the disease. After all that has been said, it can be easily understood that, while in a certain class of cases the period of incubation will be one or two days, in another class ten or twelve days will have elapsed ere this stage is completed.

In regard to the outbreak of first, or apparently first, cases in an epidemic, air polluted by bad drainage, or leaky sewers, have been accused in diphtheria as well as

in typhoid fever and in dysentery. Not only the impairment of general health, but the direct and unmistakable disease has been attributed to it. Thus Bayley refers, in the endemic of Bromley (Sanit. Record, Aug. 10th, 1877), the first cases to unventilated sewers and cesspools. School-children multiplied the disease. Thursfield attributes the diphtheria at Ellesmere (San. Rec., 158, 1877) to the accumulation of excrements under the schoolroom, and to deficient supply of water, which moreover was of bad quality. Tripe (like Railton, Bailey, Russel, Bell) accuses sewer gas (San. Rec., June 14th, 1878); others, polluted waters or bad drainage (April 18th, May 2d, 1879).

In regard to polluted water, I do not think that pathologists who attribute infectious diseases to bacteria only, are justified in condemning it. It may not be so guilty after all. For the admixtures, anorganic and organic, minerals, admixtures of wood and plants, also lower fungi and their products—algæ, infusoria—would render water rather disagreeable, but not exactly unhealthy. The latter effect can be accomplished—always assuming the bacteria theory correct, for the sake of argument—by bacteria only. But when they arrive in the stomach, their doom is sealed, they are decomposed. The only places where possibly they could take root would be diseased or ulcerated places in either the oral cavity or the upper portion of the œsophagus. Filtering would be of no use, if it were advised for hygienic purposes, for bacteria pass any filter except clay. The plea of taste or appearance alone is not sustained before the bacteria forum, the problem of diphtheria is not an æsthetical question* at all.

* The principles and the method of the microscopical examination of water have been discussed by L. Hirt, in *Zeitsch. f. Biol.*, 1879, Vol. 15. He distinguishes and treats separately of three varieties and classes of water; 1st, pure water; 2d, suspicious; 3d, putrid water, not fit to drink.

No. 1 contains no organisms even after three or five days; a few algæ or diatomacæ are permissible. They live on anorganic material only and do not change the character of the water. Some bacteria are almost always found.

Not only water, but milk also has been found guilty of giving rise to diphtheria endemics. According to W. B. Power,* we already know of certain diseases in the cow capable of affecting the human subject with disease. A. There is vaccinia, a disease of which we know that it belongs to the same class with small-pox. B. "Foot and mouth disease," which in its various stages affects the milk secretion to a varying degree, and is transferable, though not readily, with the milk, giving rise to apthous affections and disturbances of the stomach and bowels. C. Miliary tubercle of the cow, which can give tubercle to animals (perhaps including man?) that consume milk of the affected cow. D. There is an anthrax fever of oxen and cows, which has been convicted of causing malignant carbuncular disease among people who have eaten the flesh, and a throat disease very much like diphtheria among pigs that have been fed on it and on the milk.

No. 2 contains saprophyta (*sphærotilus natans*, *leptothrix*, *anthophysa Mulleri*) which live on the products of putrefaction, and larger infusoria. Also hair, wool, wood, as accidental admixtures.

No. 3, large masses of bacteria, saprophyta, and infusoria. A turbid appearance of water does not prove the presence of organic impurities; they may be anorganic, iron, etc. Moderate quantities of all of the above parasites are of no account, however. But the most dangerous inhabitants of water are large numbers of flagellata, which live on dissolved organic material and may be looked upon as the main putrefaction infusoria (*monas*, *chilomonas*, *peranema*, *englema*).

Dr. Langfeldt (D. Viertelj. f. öff. Ges., XII., 1880, p. 522) experimented in regard to drinking water containing microscopical animals, which are not destroyed by tea, coffee, Selters water, effervescent draughts, or a mild admixture of alcoholic beverages. He observed *nais*, *proboscidea*, *anguillula fluviatilis*, *euplotes charon*, *oxytricha gibba*, *zoothamnium nutans*, *colpoda cucullus*, *volvox globator*, *loxophyllum lamella*. The addition of one-twentieth per cent of citric acid destroyed all of these within two minutes. When dead they sink to the bottom of the vessels within a minute, and can be avoided in drinking. It is desirable to dissolve the acid each time before using, as a solution is liable to spoil when preserved. Only such as are protected by a harder skeleton—*cyclops quadricornis*—or a thick epidermis were not affected by the citric acid solution.

* Thoughts on the Nature of certain observed Relations between Diphtheria and Milk. Trans. Path. Soc., London, XXX., 1879, p. 546. Brit. Med. Journ., Jan. 11th, 1879. Practit., 1879, XXII., p. 306.

Mr. Powers concludes that, therefore, though a connection between diphtheria and the consumption of milk have not been proven as yet, still it is very probable indeed. His careful investigations into the causes of some local epidemics in North London exclude any other source from which the people could have been affected. Perhaps one of the forms of "garget," cow mammitis, is of an infectious character.

His reasoning, however, is not accepted by A. Dowrus,* who still believes that the milk, which gave rise to diphtheria at a distance, may have been soiled and infected. For though the connection between milk and scarlatina and typhoid fever be known for years and variously studied, no observation of the kind had yet been made in regard to diphtheria. Besides, where the young, in England, drink much milk, viz., in the cities, diphtheria was very much less frequent than where little or no milk was taken, viz., in the country. Even in the country, the well-to-do classes, who drink milk, had but little diphtheria, while the children of the poor, who obtained none, suffered a great deal from it.

In regard to this transmission of diphtheria by means of milk, O. Bollinger† hesitates to express any opinion except that the matter is very doubtful indeed. For the mode of the infection of the milk is certainly obscure, perhaps more so than the connection of typhoid fever with milk. "Garget" being a name for different conditions of the udder, a differential diagnosis will be required in order to determine which form of garget is so diphtheritic as to transfer diphtheria. This latter disease, however, does not appear to be very uncommon amongst the bovine race, provided the epidemic malignant catarrhal fever is of that nature. It is mainly found in the nares, mouth, and larynx.

Probably the possibility of contracting diphtheria directly from animals is very much greater than the danger

* Diphtheria and Milk Supply. Brit. Med. Journ., Feb. 1st, 1879.

† D. Z. f. Thiermed. u. vergleich. Pathol., VI., 1879, p. 7.

from water or milk. If that be so, many obscure cases, endemic or epidemic, will admit of a readier explanation than at present.

On a Pomeranian farm, during the winter 1875-6, every newly-born calf died of a disease with the following symptoms: More or less abundant salivation, yellow or greenish discharge from the nostrils, swelling of the cheeks, cough, and sometimes diarrhea. Extensive ulcerations of the cheeks, tongue, palate, nasal fossæ, larynx, bronchi, and intestines; ulcerations about the feet, caseous deposits in the lungs, with a beginning of pleurisy. In the liver, spleen and kidneys, nothing abnormal. The ulcerations presented the character of diphtheritic ulcerations: plenty of micrococci on the surface, inside a network of fibrinous filaments, inclosing round cells, micrococci, and detritus of mucous membrane. A healthy calf was affected the same way after having been brought in contact with the diseased ones. New-born lambs were infected by inoculation. Not so grown-up cows and bulls. But the superintendent of the farm and the woman who attended the calves were taken with diphtheritic angina.* A number of similar observations were made by F. Blazekovic.† He, moreover, like Damman, unlike Bugnion‡ counts the epidemic catarrhal fever of the bovine race amongst diphtheritic affections. Observations of his own prove contagiousness, mainly of the newly-born calves, the effectiveness of isolation, and infection of the lungs. Congestion of the bronchial mucous membrane and local pulmonary congestion he also found in calves that still were in apparently good health. It is not evident from his remarks whether he believes in a local or constitutional origin of the disease. The incubation lasted five days or less.

In a short paper on mycotic diseases in birds, O. Bollinger reports a mycosis of the trachea and lungs. The bird, a par-

* Damman: D. Zeitsch. f. Thiermed., 1876, p. 1.

† The same, 1878, p. 64.

‡ Same Jour., p. 87.

rot, died of croup of the trachea, the obstruction resulting from a clot consisting of accumulations of *aspergillus glaucus*, fibrin, and corpuscles. Another case revealed a similar affection of the bronchi with *aspergillus nigrescens*, and a mycotic, ulcerous endocarditis of the valves of the left heart in a hen. The endocarditic deposits consisted of zooglœa, coagulated fibrin, and a certain number of red and white blood-cells. There was besides, œdema of the lungs, tumefaction and fatty degeneration of liver, with yellowish infarctus in one lobe, spleen of double size, kidneys enlarged, hemorrhagic enteritis, universal anæmia, numerous micrococci in liver, spleen and kidneys, and in the blood. In the same yard, fifty fowl had died within a short time.*

Friedberger's report presented to the Veterinary Society of Munich,† on croup and diphtheria of domestic fowl leaves no doubt as to its frequency, particularly amongst the nobler varieties. The mouth, throat, and larynx (with croup symptoms), nares (serous, purulent, fetid discharge), cella infraocularis (which takes the place of antrum Highmori), conjunctiva, cornea, sclera, upper portion of intestinal tract (catarrh), are affected as they are in the human patient, perhaps with a greater liability to destruction of the eye—while the fever, also like what we see in the human patient, does not always appear to be high. Leucocytes in large numbers, detritus at an early period, pavement epithelium in small numbers, many bacteria, micrococci, and bacilli, also fungi of a high order, but little fibrin, were usually found. But there were many specimens of deposits taken from the *cavities* (cella infraocularis, etc.), *where no bacteria were found*. The course of the disease is slow, generally extending over several months, the mortality great mainly amongst the finer varieties of pigeons

* Z. f. Thiermed., 1878, 253.

† D. Z. f. Thiermed., V., 1879 p. 161. Friedberger refers to Dupart 1868, Peroncito 1870, Unterberger 1872, Siedamgrotzky 1872, Buhl 1877, Saur 1875, Reul 1874, Wortley 1877, Konhäuser 1878, Greuter 1878.

and hens; it amounts to eighty per cent, and the treatment is very unreliable.

Nicati * studied an epidemic diphtheria amongst hens, which had similar symptoms and a course very much like that in man; it could be inoculated into other animals, and was cotemporaneous with the outbreak of the epidemic amongst the human population of Marseilles. Trasbot † succeeded in inoculating a healthy hen from a diphtheritic one, but the attempts at transmission to dog, pig, and man were unsuccessful. The Med. and Surg. Journal (Med. Rec., Nov. 8th, 1879) contains the following: In a house at Ogdensburgh, N. Y., five children were ill with diphtheria. Three kittens who had been playing with them from time to time, took the disease and died. Post-mortem examination showed diphtheritic membranes in their throats.

SUMMARY.

Diphtheria is very contagious. Both the patient and his surroundings, dwelling, furniture, towels, etc., convey the disease. In dwellings it rises to the upper stories with the current of warm air. The poison clings mostly to mucous membranes. Mild cases may communicate serious ones and *vice versa*. The period of incubation lasts two days or more. It may last a fortnight. Fresh wounds do not require so long to be affected. In these cases the supposition is, that the patient was already influenced by the epidemic. Visible symptoms of diphtheria are often noticed after the constitutional ones.

* Revue d'Hygiène et de Police Sanitaire, 1879, p. 3.

† De la transmission de la diphth. des animaux à l'homme. Gaz. hebdom., 1879, avril 25.

CHAPTER V.

SYMPTOMS.

PHARYNX AND SOFT PALATE.

In the majority of cases, the disease has a prodromal stage of longer or shorter duration; usually it lasts a day or two, and may run a similar course to that of a catarrhal pharyngitis. The patient feels somewhat indisposed, has slight fever, is dejected, complains of painful deglutition, more marked when swallowing fluids than solids or semi-solids, has headache and, occasionally, vomiting. The occurrence of the latter, however, is very much less frequent than in the outbreak of scarlatina. In very severe cases, convulsions have been observed; chills very rarely; elevations of temperature of from 102.5° to 104° F. are frequent; higher ones, from 105° to 107° , seldom occur. At this time it is often difficult or impossible to distinguish a catarrhal angina from a diphtheritic, by the subjective symptoms. Slight glandular swellings under the jaw may occur in either. The characteristic objective symptom of the latter disease is the presence of membrane on the reddened mucous membrane of the fauces, which is markedly injected over all or part of the surface. The arches of the palate and the tonsils, less frequently the posterior wall of the pharynx are so affected. A distinctly localized redness cannot be but either traumatic or diphtheritic. Larger or smaller deposits are found thereon, lying loose on the surface or deeply imbedded according to the locality. At times the first examination reveals their presence in large numbers, at other times but a single one can be detected, which is soon followed by others, however. Within a certain period of time, as a rule twelve to twenty-four

hours, the single deposits coalesce and form a membrane of greater or less extent. Mostly in the same proportion to its increase in size, it increases in thickness, partly by an actual homogeneous growth, partly by the addition of blood, mucus, or other foreign substances. On the uvula, soft palate, and the posterior wall of the pharynx, the membrane is located superficially and at times can be easily removed; on the tonsils, it has a firmer hold and is occasionally amalgamated with the uppermost tissues thereof. On the other hand, there are cases in which no actual membranous formation occurs; in such cases the tissues are more or less swollen, the surrounding portions more or less reddened, and the grayish-white discoloration is the result of an infiltration of the tissues themselves, and cannot be removed, as was possible in the cases previously spoken of.*

There are still other cases, in which deposits of membrane and tissue infiltration are found at the same time, and where both history and evidence indicate that these two phenomena are the result of one and the same process. When the uvula takes part in the process, the swelling is, as a rule, more marked than when the remaining parts of the fauces only are implicated. Its circumference is very considerable and amounts sometimes to the treble or quadruple of the normal, in consequence of the œdematous condition of the entire tissue.

We have to deal, then, with three different manifesta-

*The diagnostic differences between primary pharyngeal diphtheria and scarlatinous diphtheria, such as O. Heubner describes them (*Jahrb. f. Kinderheilkunde*, XIV., p. 1), are in my opinion very much overdrawn. According to him, they differ both clinically and anatomically. In scarlatina the deposits are always thin (?), no membranes are thrown off (?), and it is impossible to remove them by artificial means (?). In pharyngeal diphtheria there is no epithelium imbedded in the membranes (?), in scarlatinous diphtheria it was so found several times, though in a changed condition. The latter gave rise to actual necrosis of the tissue, and in several cases to general pharyngeal gangrene. Very few of my readers will admit that the views of the writer correspond with their own experience to such an extent as to induce them to obliterate in their minds the various transitions between the extreme forms above depicted and to admit their essential difference.

tions of the diphtheritic process: first, with a membrane lying on the mucous membrane, and removable without causing much injury to the epithelium, or any to the basement membrane; *such membranes were given by some the name of croupous deposits*; secondly, with a membrane implicating the epithelium and upper layers of the mucous membrane; to this, the *title of diphtheritic membrane has been given*, by preference; thirdly, with a whitish or grayish infiltration of the surface and the deeper tissue, which, if abundant, may give rise to a necrotic destruction of the tissue.

The severity of the disease does not always depend on the predominance of one of these three forms, for any of them may accompany a mild or a severe attack. By a severe attack we understand one attended with chills, temperatures as high as 105° and 107° F., and marked nervous symptoms, such as vomiting and convulsions. It is characteristic of such cases that, when the membrane is accidentally or forcibly removed, it is speedily reproduced; the lymphatic system, in addition, takes an active part in the process. The neighboring glands become swollen, the periglandular tissue does likewise, so that the circumference of the neck becomes enormous, and the space between the lower jaw and the clavicle appears one immense tumefaction. These are the cases in which, as a rule, loss of strength and general debility speedily ensue, and death occurs from exhaustion. The membrane, in cases of this description, frequently undergoes changes in appearance; under the influence of the atmosphere and of foreign substances, and by admixture of blood, its color becomes yellowish or brownish. The odor of the membrane and surrounding parts becomes sweetish and musty, and occasionally so fetid that it contaminates the atmosphere of the room, and the air in its transit through the nose and over the pharynx becomes by inhalation still more infectious and dangerous to the patient. His throat becomes more swollen, his respiration loud, he keeps his mouth open constantly, has an indifferent expression, the saliva dribbles

continually, the color of the skin is sallow and livid, the appetite very poor, and pulse both frequent and small. When the symptoms are of long duration, and a deep infiltration of the affected parts occurs, hemorrhages not infrequently make their appearance. These may be slight although frequent; occasionally, however, larger blood-vessels are encroached upon in the process of destruction, and dangerous, nay even fatal hemorrhages may be the result. The septic forms which I have here described are more dangerous than the mild ones, previously mentioned. Still, even in the latter, bad results may ensue from a direct absorption into the blood of putrid substances, and by the penetration of fetid gases to the lungs.

Occasionally, where the infiltration has been extensive, we meet with a condition that can only be considered as gangrene. In such cases we see collections of a grayish pulpy mass which, on falling off, leaves a considerable loss of tissue; the further course of the disease being either favorable, or dangerous through absorption of septic material, or accompanied by local hemorrhages. When after a time the health is completely restored, marked cicatrices are left behind. Such loss of tissue is generally seen in the tonsils only, but it may also be encountered in the soft palate. Its cicatrices on the soft palate are always a source of inconvenience, partly in swallowing, partly in speaking. Actual local perforation of the soft palate I have seen but four times in more than twenty years, sloughing without perforation, very often.

NASAL CAVITIES.

The diphtheritic membrane not infrequently spreads from the pharynx to the neighboring organs. From the posterior aspect of the soft palate or pharynx, the disease gradually ascends to the nasal cavities; this is particularly apt to occur when the uvula is the seat of extensive deposits, and by forced inspiration and deglutition its poste-

rior surface becomes affected. In such cases, the membrane which extends thence to the nasal cavities is very dense, and capable of narrowing the capacity of the nasal cavities anteriorly, and occasionally even to close them entirely; as a rule, however, several days elapse before the membrane assumes such a condition. Usually, when this form of nasal diphtheria is in its incipient stage, it is impossible to diagnosticate it; the most important sign thereof, beside a more nasal articulation, and sometimes greater difficulty in deglutition, and the result of close ocular examination while the uvula is turned sideways or drawn forward, is a swelling of the deep facial glands at the angle of the lower jaw; when these swell rapidly, it can be asserted positively that the nasal cavities have been invaded. Frequently there is little or no discharge from the nostrils under these circumstances.

The picture is a very different one, however, when the nose becomes primarily affected. This usually occurs only where an acute catarrh with but little secretion, rarely where a chronic catarrh has immediately preceded infection. When the secretion was thin and serous, the diphtheritic infection renders it no thicker, but makes it slightly flocculent, and it may become very profuse. This form is frequently attended with a disagreeable odor, equally unpleasant to the patient and to those around him. During the prevalence of an epidemic, one must always be prepared to see an acute nasal catarrh, or an influenza, or even a chronic nasal catarrh become complicated with diphtheria or pass into it. Schuller reports the case of a five-week-old male child who, having had a nasal catarrh since birth, became affected with diphtheria of the nose. The glandular swelling of which I spoke above is a very important diagnostic, and likewise a decidedly unpleasant, symptom which becomes very marked inside of twenty-four hours; frequently a partial swelling remains long after the disappearance of the diphtheritic membrane. Such glands rarely suppurate or undergo a necrotic degen-

eration; a permanent induration thereof, however, not infrequently occurs.

A chronic catarrh of the pharynx (and larynx), also the nose, is apt to remain after a serious attack of diphtheria. In that respect the latter does not differ much in character, though sometimes in degree, from attacks of non-infectious inflammations; they also are the starting-points for a number of subacute relapses or for chronic ailment. Œdematous swelling of the mucous membrane and submucous tissue is often observed for a long period to come; elongated uvulæ, enlarged tonsils, often date back to such an acute attack. Thus it is with the upper portion of the larynx about the posterior insertion of the vocal cords (see below); its large amount of loose submucous tissue is liable to swell considerably in acute attacks. Frequent spells of croupy cough, and a certain degree of dyspnœa is often observed for years afterwards. Though the cases of genuine cicatrization between the arytenoid cartilages, as described by Michael (*Deutsch. Arch. f. klin. Med.*, 1879, XXIV., p. 618), be rare, with their result of permanent paresis of the thyro-arytenoid interni muscles, still the cases are very obstinate.

EYE.

From the nose, the diphtheritic process frequently extends to the nasal duct and from thence to the conjunctiva of the eye, although it is not claimed that diphtheritic conjunctivitis originates in this way only. In the early history, especially of the epidemic that has prevailed for the past twenty-two years, I frequently saw diphtheritic conjunctivitis either as a complication of diphtheria of the fauces or nose, or occurring primarily. Diphtheritic conjunctivitis is an exceedingly dangerous disease as far as the safety of the affected eye is concerned; not infrequently the cornea is destroyed within twenty-four hours by gangrene from pressure, or by diphtheritic keratitis. Diphtheria of the conjunctiva is not, fortunately, a very

common affection. It appears to be more frequent in some epidemics than in others. At all events, I have seen many more cases, compared with the whole number, in the first four or five years of the prevailing epidemic than in the last seventeen or eighteen. Its symptomatology and pathology were first studied by A. von Graefe, in 1854, but have since been the subject of frequent and careful observation. The upper (sometimes the lower) eyelid is first taken, becomes suddenly red and rigid, and is greatly swollen. But rarely a single eyelid is the only seat of the affection during the whole course. At first the conjunctiva palpebrarum is smooth, dry, and pale, while that of the eye is chemosed, afterwards diphtheritic membrane is deposited in floccules or solid masses. After a few days the deposit begins to macerate and the eyelid is less hard. Deposits on the conjunctiva bulbi are less hard, and peel off more easily. At an early period, that is, often within twenty-four hours, the cornea becomes hazy and ulcerated. Perforation is very apt to take place, and either prolapse of the iris, or destruction of the eye, at all events loss of sight, are of frequent occurrence.

EAR.

In the same manner as the eye, the ear may become affected by continuity with the naso-pharyngeal space. The orifices of the Eustachian tubes in children are narrower than in adults, and present a slit-like aperture; hence a slight swelling of the mucous membrane or a moderate diphtheritic deposit may close them, and hardness of hearing be the result. In these cases, the patient not infrequently complains of intense pain behind the angle of the jaw and in the ear. In some cases, the diphtheritic membrane is continued into the tubes, giving rise to otitis interna and media, and finally terminating in a perforation of the drum-membrane, and occasionally in caries of the bones of the ear.

Moos describes a case of diphtheria of the external audi-

tory canal in a boy of ten years, who had frequently suffered from a purulent discharge from the middle ear. He observed a profuse exudation holding fast for a number of days and accompanied during detachment by pain and hemorrhage; no complication existed in the throat or elsewhere. P. A. Callan (*Med. Rec.*, 1875, p. 221) reports a similar case. Gruber asserts that diphtheria of the external auditory meatus occurs only when it has for some time previously been the seat of a catarrh. I recollect but one such case in my own practice, and but one primary case in which no catarrh preceded the diphtheritic membrane. Bezold, however, publishes (*Virch. Arch.*, 70, p. 329) three observations of fibrinous exudation on the drum-membrane and in the external ear. Wreder (*Montassch. f. Ohr.*, No. 10, 1868) collected eighteen cases of diphtheria of the middle ear in scarlatina, complicated with the same affection of the fauces and nares; one child with diphtheria of the mouth and pharynx had also diphtheria of the inner ear. Küpper saw diphtheria of the cavum tympani and Eustachian tube; Wendt, once in the tubes, and twice, in eighty-four cases of variola, in the middle ear, together with naso-pharyngeal cavity (*Thierfelder Atlas histol. Anat.*, tab. I.). Wreder saw a case of primary diphtheria in the external ear.

EPIGLOTTIS, LARYNX, AND TRACHEA.

The descent of the diphtheritic process into the respiratory organs may give rise to various conditions. The membrane is not always found to pass uninterruptedly from the mucous membrane of the fauces into the larynx; not infrequently isolated diphtheritic spots are found in the pouches on either side of the attached extremity of the epiglottis. At such times, the epiglottis is moderately swollen, its margins hard and reddened. Occasionally the redness is interrupted by small diphtheritic deposits which may remain isolated for a considerable time, but finally coalesce so as to coat the edges of the epiglottis with a

continuous membrane. As a rule, the upper surface of the epiglottis is not completely covered by membrane, while only now and then diphtheritic deposits are found on its under surface.

A most extraordinary case of diphtheria of the epiglottis I have seen in company with Dr. Wm. Balser. A girl of four years was attacked with pharyngeal diphtheria of rather a mild type, the general condition being fair, and fever moderate. In due time mild symptoms of croup set in, moderate hoarseness, dyspnœa, and croupy cough. The epiglottis was a little hyperæmic, but not all over. On some hyperæmic spots, but also on those of normal color, diphtheritic deposits would show themselves, but the appearance of a hyperæmic spot would not necessitate a subsequent diphtheritic membrane. The deposits were never large, never at any time covered so much as one-third of the margin of the epiglottis at the same time; their first appearance being on the left, it took more than two weeks before this spot got clean again; meanwhile, a few isolated little deposits took place on the centre and to the right, the disappearance being as slow as the development. The whole process lasted a month in this way, the croup symptoms being well pronounced, but of a mild type.

The subjective symptoms accompanying the affection of the epiglottis are not always in direct proportion to the extent of the membranes. Dyspnœa and hoarseness occasionally occur where the only abnormal condition is a marked œdema at the entrance of the larynx, particularly of the posterior wall near the arytenoid cartilages and the attachment of the vocal cords. The œdematous condition causes a functional paralysis of the vocal cords, together with marked dyspnœa on inspiration. The difficulty of breathing may become so excessive that the clinical diagnosis of croup is unquestionable, and tracheotomy resorted to, while expiration is comparatively free, and the voice not markedly affected. Furthermore, cases occur in which there is no marked œdema, but merely a general catarrh of

the epiglottis and larynx; here, too, the subjective symptoms of hoarseness and dyspnoea may become severe, and necessitate the performance of tracheotomy. Still, bearing this in mind, I have on several occasions refrained from performing this operation, where I judged that, aside from the diphtheria of the pharynx, I had to deal with a moderate oedema of the glottis or a laryngeal catarrh.

Frequently, however, membranes form in the larynx in the same way as in the pharynx or nose; then inspiration and expiration are equally interfered with, and hoarseness is a more constant symptom than in the above-mentioned cases. Fever and pain are not necessarily prominent symptoms; in fact they are frequently unimportant, but in proportion as the degree of narrowing of the larynx increases, the respiration becomes more difficult, long-drawn, and loud, in the place of hoarseness. There is complete aphonia, and the hoarse, loud, barking, "croupy" cough becomes more husky and suppressed, without losing all of its croupy character. This narrowing may continue and increase for days, when suddenly a paroxysm of severe dyspnoea comes on. The child can no longer lie down, becomes pale, livid, and bluish, its skin is covered with perspiration, it supports itself on its hands and knees. The inspiratory act is slow and whistling, expiration short and shallow, pulse becomes intermittent, and as the condition of carbonic acid poisoning comes on, convulsions ensue from time to time. Such a paroxysm sometimes lasts considerable time, leaving the patient in a worse condition than before the attack. It is immaterial whether the dyspnoea be sudden or gradual; in either case the respiratory muscles have an unusually severe task to perform. The supraclavicular and intercostal regions are retracted with each inspiration, and in severe dyspnoea the ensiform process takes part in the exhausting respiratory efforts. Long before this, the vesicular murmur cannot be heard on auscultating the lungs, for the normal respiratory sound is masked by the laryngeal noises, even though the lungs

had not as yet been directly implicated. Paroxysms may recur again and again, invariably leaving the patient in a worse condition. Occasionally the child dies in such an attack, at other times the paroxysm appears to bring about a state of comparative comfort, the child remains more or less cyanotic, becomes oblivious to surroundings under the anæsthetic influence of the carbonic acid poisoning, and finally dies of asphyxia. Anæsthesia, however, is not so frequent as some writers appear to believe. This is the picture of membranous croup, as seen during the long and wide-spread epidemic of diphtheria, that has lasted up to the present day, these twenty-two years.

As a rule, a number of days elapses between the appearance of the first laryngeal symptoms and death; at times, however, the laryngeal symptoms appear very suddenly, and may lead to a fatal result in a few hours. This is possible where there has been a very large formation of membrane within the larynx, but it usually occurs only when the trachea and bronchi were the first to become the seat of diphtheritic deposits, the larynx escaping infection in the beginning. It may happen that the trachea and bronchi may become affected, although diphtheria of the fauces do not exist. This does not occur as rarely as Henoch and Oertel seem to believe. The former thinks that *diphtheritic tracheo-bronchitis* is taken to be the primary condition, because the throat is not examined early enough. He gives his opinion on a case (Charité-Annalen, Berl., 1876) in the following words: "On examining the throat of a four-year-old boy, upon whom Henoch had performed tracheotomy, November 20th, 1873, nothing further than redness could be detected, and on autopsy there was naught but hyperæmia of the velum, tonsils, and pharynx, and a swelling of the tonsils. But low down, underneath the base of the tongue, on either side of the epiglottis, the mucous membrane was covered by diphtheritic membrane, which spread from thence far down into the bronchial tubes." Henoch's statement in refer-

ence to this case is lucid enough, but the above-described condition allows of another interpretation. If the case was of long duration, it is probable that Henoch's theory, that the throat was not examined early enough to observe the downward passage of the disease, is appropriate. But the cases that run a rapid course, and the invasion of which is akin to a thunderbolt, are those that began below and extended upward.

Oertel is of the opinion that the membrane in the fauces is overlooked in such cases. Steiner, too (*Ziemssen's Handb.*, IV., 1, 236), thinks that "the tendency of the times is to question, nay, rather to deny, the existence of croup extending from below upward." Now, on the contrary, repeated experience enables me to assert with positiveness that diphtheritic tracheo-bronchitis may occur without an affection of the pharynx at the same time. I do not deny that it may last for days without giving rise to dangerous symptoms. I know it does. But when the process reaches the larynx, the symptoms of suffocation become so urgent that tracheotomy may be absolutely required at once, and, in spite of the operation, death soon after occurs.

It is characteristic of these cases that when, immediately after tracheotomy, a feather be introduced through the tube, the contact thereof with the trachea below the extremity of the tube will not give rise to coughing. In about fifteen such cases, or more, I have performed tracheotomy on, I have seen the first speck appear in the pharynx the same day, or soon after; and, where the children lived long enough, say twenty-four hours after the operation—a rare occurrence, though—the speck had spread into a large membrane. In one case in which I performed tracheotomy, and no membrane and but slight swelling of the fauces could be detected during life, an autopsy revealed no deposits in the fauces, but the larynx, trachea, and several ramifications of the bronchi contained diphtheritic membranes. Nor is it possible that, when death

ensues rapidly from suffocation, the process should have a sufficient time to spread upwards to the pharynx.

Of course, these cases are exceptions; as a rule, laryngeal and tracheal diphtheria result from a descent of the disease from the fauces. More or less uncomplicated cases of primary laryngeal diphtheria, or so-called sporadic membranous croup, were, however, observed before the end of the sixth decade of this century. They were then almost the only cases of diphtheria, and linked former epidemics and the present one together. Further explanation of this subject will be found below, in my anatomical remarks.

INFLAMMATORY AFFECTIONS OF THE LUNGS

may occur at various times and in various forms during an attack of diphtheria. That which appears after tracheotomy is usually a broncho-pneumonia, the result of rarefaction of the air in the respiratory passages during the period of impeded inspiration, with consequent collapse of pulmonary tissue and dilatation of the blood-vessels, and hence a disturbance of the circulation; it may not fully develop until after tracheotomy, and is a frequent cause of death on the second or third day succeeding the operation. Now and then a case of lobular pneumonia will result from the aspiration of pieces of membranes into the smallest bronchi. It can be easily recognized when the trachea is opened, but previous to the operation the auscultatory signs are of little or no value, being masked by the laryngeal râles. Percussion is equally useless, for a dulness might just as well indicate collapse of the lung as infiltration. The second form of pneumonia associated with diphtheria, whether of the pharynx or larynx, is from the beginning fibrinous in character. Here, too, auscultation and percussion are of little assistance in establishing a diagnosis, when there is a laryngeal diphtheria at the same time, for the above reasons. Where, however, the dulness on percussion is accompanied by

high fever, and the long-drawn inspiration is replaced by rapid respiratory movements, we have reason to think of pneumonia as a complication.

Roser and Pauli believe that pneumonia may result from the entrance of blood into the lungs during tracheotomy; this is denied by Steiner. The former view this question from the same stand-point as that taken by Niemeyer when he imputed to hemorrhages in the minutest air-passages the power of bringing on inflammatory changes in the respiratory apparatus. I am in a position to testify to the truth of his remarks, from having myself witnessed such a phenomenon. Having performed tracheotomy in a healthy, ten-month-old child for a foreign body in the air-passages, the child died on the operating table an hour after the opening of the trachea, before the necessary attempts at extraction had ceased. A number of the smallest bronchial tubes contained blood, which had flowed in during the protracted operative proceedings; around these collections, the lobuli were collapsed and pale. No doubt these spots would have become the seat of fluxionary hyperæmia and inflammation had the process been continued, unless a removal of the blood-clots had ensued, which I should consider very improbable indeed.

DIPHTHERIA OF THE MOUTH,

as a primary affection, is not of very frequent occurrence; not rarely, however, is it associated with diphtheria of the fauces and nose, mainly when they have assumed a septic or gangrenous character; it appears on cheeks, tongue, angles of the mouth and gums, and after the fetid discharges have excoriated the skin, on the lips also. In all of these localities it appears less in the form of an extensive, thick membrane than an infiltration of the tissues, to wit: in and under the mucous membrane, in the accidental fissures of the tongue and corners of the mouth. It is most apt to occur where, from the start, the mucous membrane of the mouth presented a solution of continuity. The ulcerated

base of a follicular stomatitis is very frequently the starting-point of a general diphtheria of the mouth. It is always a disagreeable symptom, points to a long duration of the whole process, and threatens septic absorption.

ŒSOPHAGUS AND THE CARDIAC PORTION OF THE STOMACH

are the seat sometimes of very massive and extensive, mostly fibrinous exudations, in typhoid fever, dysentery, cholera, measles and scarlatina, or after injuries following the contact with mineral acids, alkalies, corrosive sublimate, or antimony. When the normal tissue was not injured, I never saw any that were not superjacent and could not easily be peeled off ("croupous"). Rokitsansky reports, however, cases of genuine infiltration of the tissue ("diphtheritic"). Most of these exudations are whitish or grayish-white. Addition of hæmatine is apt to give it a brownish admixture. A case of L. Letzerich's ("mykosis œsophagi," in *Arch. f. Pathol. u. Pharmac.*, VII., p. 33) does not belong here, probably. A girl of one and a half years had now and then severe pain in swallowing, had no appetite and no sleep; after a few days, stomach large and tympanitic, no fever, not much vomiting. Nothing in the pharynx. The pain during deglutition finally pointed to the œsophagus. Œsophageal epithelium (none from the stomach), broken up and degenerated, also micrococci are reported to have been found. It was also learned that the baby had picked off and eaten rotten wall-paper. The locality of the affection could be explained only by assuming that there was, previously to the passing down of the micrococci to the stomach, where they would have been decomposed and rendered harmless, a sore spot met with in the mucous membrane of the œsophagus. But the following case is a better illustration of what I mean. I met in a three-year-old child in a tenement house in Thomas street, some eighteen years ago, during well-developed typhoid fever, with a fibrinous exudation, beginning in the pharynx

and filling the whole of the œsophagus, the cardiac portion of the stomach being also covered with a slight film. Dr. W. S. Greenfield presented to the London Pathological Society (Trans., vol. XXIX., 1878, p. 29) the case of a female child, aged five years, who during life and at the autopsy exhibited all the symptoms of enteric fever, and also on the tongue, throat, larynx, and the upper part of the trachea, the false membrane of the usual diphtheritic character.

According to Zenker and v. Ziemssen, the membrane of pharyngeal diphtheria is said to suddenly cease at the entrance into the œsophagus. This has not been so in my experience. The upper part of the œsophagus, over a surface of one to three centimetres (one-half to one inch), is often covered in cases of extensive pharyngeal and laryngeal diphtheria. The upper portion of the deposit is very much like the adjoining pharyngeal membrane, but the lower part is thinning out into a mere film, soon. I found it to be so in many autopsies of membranous croup. On the other hand, I met with a case in which the diphtheritic deposit was found in the lower part of the œsophagus only. It was that of a boy of four years who suffered from an almost impenetrable stricture of the œsophagus (produced by his swallowing lye a year previously), when he was admitted to my service in the Mount Sinai Hospital. Careful dilatation was resorted to, which was interrupted frequently and just as often recommenced, until he began to partake of solid food. After a period of what appeared to be a common bronchial catarrh, attended with but occasional fever, serious dyspnœa set in, and the child died after a brief attack of fibrinous tracheo-bronchitis. The usual results of the post-mortem examination of the respiratory organs were not even so interesting as the condition of the œsophagus. Its entire length was normal, with the exception of a circular portion about three centimetres in length, commencing about twelve centimetres (five inches) from the upper end. It proved absolutely im-

penetrable to the smallest silver probe, while but a few days previously the patient had swallowed semi-solid food. The cause of the obstruction was found to consist of a deposit on, and infiltration into, the cicatricial tissue. Now, cicatrices are not very apt to be affected unless their surface is eroded. It is highly probable that the difficulty of deglutition, and the frequent use of the bougies, prepared the œsophageal stricture for the diphtheritic deposit which has been alluded to.

INTESTINAL DIPHThERIA.

My experience concerning *intestinal diphtheria* is not large. Of course, I except dysenteric affections. In one case, that of a three-year-old boy (a fatal case of diphtheria of the throat had occurred in the same house the year before), the symptoms were fever, moderate tenderness of the abdomen without much tympanites, constipation, and great prostration. The diagnosis was enteritis. An autopsy revealed diphtheritis, having its seat in the jejunum and ileum. The membranes consisted of a dense network with granular contents and but slight intermingling of mucus. In the cow, croupous enteritis is frequent. Tubular casts of two metres in length, one of nine metres (which was passed at one time in eleven segments, and contained numerous specimens of *tænia denticulata*) have been observed (O. Bollinger, in Deutsch. Z. f. Thiermed., 1st suppl., 1878, p. 18). Weissenfels has a case of diphtheria of the gall-bladder from irritation by a calculus (Inaug. Dissert., 1868). I. Zit (Jahrb. f. Kind., 1879) found most cases of intestinal diphtheria in the large intestines, not counting the cases of dysentery, which in his experience was often met with in complication with pharyngeal diphtheria. Still in the upper portion of the intestinal tract diphtheritic enteritis was by no means rare. The deposits were quite thin, sometimes, but were characteristic in their composition. Still I warn against a mistake which can easily be made unless the microscop-

ical examination of the long tough membranes be resorted to. Such membranes consist sometimes of nothing but mucus, hardened and flattened down by long compression.

WOUNDS.

Wounds of all kinds are easily and rapidly infected by diphtheria. I have already spoken of the diphtheritic infection of vaginal abrasions and also of erosions of the external ear, tongue, and corners of the mouth. Scarification or removal of part of the tonsils is followed in half a day or a day by a deposit of diphtheritic membrane on the wound. The wound caused by tracheotomy becomes infected with diphtheria within twenty-four hours. Leech-bites, skin denuded by vesicatories, removal of the cuticle by scratching during cutaneous eruptions, all furnish a resting-place for diphtheria in a short time. Larger wounds, as those of amputations and resections, may speedily become covered by diphtheritic membrane and thus easily lead to death. I have seen two cases of resection of the hip-joint in my own practice which ended fatally in a short time by complication with diphtheria. Therefore, I have made it a rule to operate as little as possible, while an epidemic is raging, particularly in the mouth.

Billroth has given the name of

MUCO-SALIVARY DIPHTHERITIS

(Allg. Chir. Pathol. u. Ther., 8. Aufl., 380) to an affection ensuing after extirpation of a large portion of the tongue, and resection of the lower jaw. The local changes consist, primarily, a in very hard and rather wide-spread infiltration of the cellular tissue about the wound, followed by its rapid pulpous degeneration. Most of these cases terminate fatally by septicæmia, some favorably by profuse suppuration starting from beneath the local necrosis of tissue. The patient is menaced by this disease only during the five days succeeding an operation, as it never occurs later than this. When healthy granulations have

already appeared, this form of diphtheria cannot occur, and should diphtheria attack the wound, it is due to a disturbance of the granulations, thereby allowing the disease to be introduced from without. The general symptoms of this affection may be very severe, and collapse ensue very early.

SKIN.

At times immediately at the beginning of an invasion of diphtheria, at other times only on the second or third day, an erythematous eruption, more or less general, appears on the skin. Now and then it appears on the chest, shoulders, and back; at other times it covers the body, and has not infrequently led to its being confounded with scarlatina. It is not always accompanied by much fever, and cannot therefore be mistaken for that form of erythema which frequently appears in children with delicate skins during high fever from any source. I cannot say that I have found this complication to give a more malignant character to the disease, but true erysipelas does. I am not prepared to prove that the two processes, erysipelas and diphtheria, are identical under some circumstances, but the complication of the two, and the ferocity with which they combine, renders a close relationship probable. I have seen an infant dying from an erysipelas added to a post-auricular diphtheria, this being due to a slight abrasion of the surface. Erysipelas originating in the tracheotomy wound, though ever so carefully disinfected and secured, is frequently observed after two or three days, and is a very ominous symptom. And erysipelatous surfaces, denuded of their epidermis by spontaneous vesication, or injured by ever so slight a trauma, are very liable to be covered with diphtheritic membranes.

DIPHTHERIA OF THE GENITO-URINARY ORGANS

does not often occur. However, I have proofs of its ap-

pearance even as a primary disease. The rarity of its occurrence, and the fact that occasionally after delivery the abrasions of the vagina, arising during labor, become covered in twenty-four hours with a diphtheritic membrane, would seem to indicate that, where diphtheria of the vagina is found in children, it is probable that the disease had its local foundation in a catarrh or erosion of that locality. Undoubted cases of that kind I have seen several times in the course of the last few years. In but few cases I have seen an infiltration of the neighboring inguinal glands; *in but a single case can I positively claim to have seen diphtheria of the pharynx following a diphtheria of the vagina*, this taking place in an adult, upon whom an operation was performed. But a single case of that description proves a great deal. I. Zit reports thirteen cases of vulvitis diphtheritica; in some the local affection was the first symptom of generalized diphtheria, with partly superjacent, partly imbedded membranes. Diphtheria of the bladder has been described by Billroth as occurring on the mucous membrane of the urinary bladder and vagina, where it is met with especially where the urine is alkaline, after lithotomy, urethrotomy, the operation for vesico-vaginal fistula, and in ectopia vesicæ. This form of diphtheria has a marked tendency toward localization, but by extension of the phlegmon, when of putrid character, to the retro-peritoneal cellular tissue, a peritonitis with fatal termination may ensue. Before this, however, can take place, and with it, sepsis from absorption may occur. The diphtheritic inflammation of the vagina probably sets up a superficial inflammation in the uterus and Fallopian tubes and may thence lead to peritonitis. Diphtheria of the vagina of a fibrinous character occasionally occurs after the operation for vesico-vaginal fistula, more frequently after delivery, when the disease usually has an unfavorable termination. I witnessed an extensive diphtheria of the vagina in a patient upon whom Dr. Emmet, at my request, performed an operation for prolapse of the vagina and extensive laceration of the cervix. The disease

commenced in the wounds, which began to gape, and from thence spread over the entire vagina. *In a few days diphtheria of the fauces appeared.* Another operation became necessary, of course, which resulted favorably. At that time there was not a single case of diphtheria in Dr. Emmet's institution, the one in question forcing him to almost empty his hospital in order to get rid of the danger. No case of diphtheria occurred subsequently. In the case of this lady, the disease appeared to have affected her alone, for, in spite of the prevalence of the epidemic, neither in her house nor in the circle in which she moved had another case of diphtheria occurred. Diphtheria of the placenta was demonstrated to the German Surgical Society (Verhandl., 1877, p. 41) after having been proven to exist by Schüller. The membrane was between uterus and placenta, and remained attached to the latter. It resulted from puerperal sepsis. Hueter also (Deutsche Z. f. Chir., 1877, p. 226) emphasizes the occurrence of diphtheria after urethrotomy and lithotomy. It may even occur after measuring stones for the purposes of lithotripsy; thus he advises that injections be made of mild solutions of carbolic acid. It is, however, natural, if not in diphtheria, still in Hueter, who insists upon "monads" to complete the diagnosis of diphtheria—that these cases of diphtheria do not "count for full," that they are "pseudo-diphtheritic." To the rabbits concerned, however, it will be satisfactory to know that inoculation of these pseudo-diphtheritic masses into their backs destroyed life as readily as that of diphtheritic membranes from the larynx. For similar observations compare also Virchow in Charité-Annalen, 1875.

I have repeatedly seen diphtheritic infection of circumcision-wounds. A Jewish family, coming to New York from the interior of New Mexico several years ago, had among its younger members three boys of from six months to five years of age. As none of them were circumcised, the rite was undertaken on all three on the same day. The wound in every one became diphther-

itic within twenty-four hours, two taking a favorable course, the third (and that in the eldest) resulting in a considerable loss of substance. In none of these cases, likewise in none of those seen previously, was there a swelling of the inguinal glands, but in the last-named, oldest boy, pharyngeal diphtheria showed itself within a few days, but healed long before the penis healed and cicatrized. Dr. Lange also (*Med. Rec.*, July 10th, 1880) saw a baby of three weeks, who had been ritually circumcised while there was diphtheritic sore throat in another child of the same family. A membrane covered the preputial surface, besides two diphtheritic patches on the scrotum.

Four years ago, I operated on a healthy boy of three years for phimosis. A simple incision through the upper, anterior part of the prepuce was made and the wound on either side closed with stitches. Mild carbolic acid applications were made immediately and permanently. Diphtheria of the wound developed on the following day. Antiseptic treatment was continued, and carefully attended to, and a number of stitches in swelled and rigid parts removed. The diphtheria extended over a part of the penis, erysipelas set in, of no great extent, however, and the child died four days after the operation.

Ad. Stromszky (*Jahrb. f. Kind.*, 1880, XV., p. 170) publishes a case of diphtheritic balanoposthitis in a boy of three years, with consecutive gangrene of the prepuce, which was complicated with lobular pneumonia and (metastatic) abscesses in both lungs, resulted in abscesses in both inguinal regions and a psoas abscess of the right side, and terminated fatally.

A boy of four years, idiotic and choreic, and suffering from a high degree of phimosis, was presented at my college clinic half a year ago. The phimosis gave rise to difficulty in micturition, and great uneasiness and pain. Therefore, but not with a view of improving either idiocy or chorea, I performed the simple operation of incising the upper aspect of the prepuce. The wound was care-

fully closed with a sufficient number of stitches, and an antiseptic application ordered. Nevertheless, in a very few days diphtheria of the wound set in, the whole prepuce and a small portion of the penis became gangrenous, and it took all the attention of my clinical assistant, Dr. Golding, and a few students through more than six weeks to save the whole organ from destruction. Considerable deformity, however, took place.

KIDNEYS.

Of the internal organs, the kidneys take the most active part in the diphtheritic process. Wade, in 1857, was the first to speak more particularly of the presence of albumen in the urine, in diphtheria. Albuminuria is not always of significance, as it occurs in severe and mild cases alike, both before and after tracheotomy, and therefore is not connected always either with the height of the fever or the degree of dyspnœa; at times it disappears in a few days, in other cases it is of longer duration. It is not invariably complicated with changes in the kidney, neither do we always discover casts or degenerated epithelial cells in the urine. In other respects, also, it does not behave like albuminuria in scarlatina. In the latter it appears seldom before the second week of the process, and frequently later, while in diphtheria it is often seen early. It sometimes lasts but a few days, particularly in many cases which set in with a high fever, which rapidly diminishes, and terminates in speedy recovery. In these occurrences the presence of albumen appears to attend the rapid elimination of the poison.

Albuminuria seldom lasts longer than a week, and is not often complicated with œdema, but sometimes it is but a symptom of a local or general nephritis, and then hyaline, epithelial, and fibrin casts, and granular cells, are found in the urine. Nephritis then assumes as serious a character as it proves to possess in scarlatina. Cases of nephritis, for-

tunately rare, in a very early period of diphtheria, run a rapid and often fatal course.

THE HEART AND BLOOD

are affected in various ways by the diphtheritic process. Where the disease runs a slow course, accompanied by high fever, a granular degeneration occurs, similar to that appearing in other acute infectious disorders, typhoid for example. In diphtheria, however, it would seem that this condition may arise even without marked elevation of temperature. The pathological changes in the heart produced by diphtheria are not always the same. Ecchymoses, cellular hypertrophy, and granular degeneration have frequently been noticed after death where the symptoms had been severe.

The result, of course, is considerable weakness of its muscular tissue, evidenced by the formation of local (Beverley Robinson) thrombi, general sluggishness of the circulation, dyspnoea, muffled heart-sounds, a cool and pale skin, and sudden death, preceded by a very feeble and frequent, sometimes, however, by a very slow pulse. Aside from this, there are actual cases of endocarditis during the course of diphtheria, or convalescence therefrom. It affects especially the valves, and among them particularly the mitral. It is characterized by high fever, precordial pain, attacks of syncope, and a systolic murmur.

In most of these cases the local deposit gave rise to the most important symptoms, but there are cases of diphtheria in which the constitutional symptoms are more prominent than the local ones. I have twenty years ago designated these cases as diphtheritic fever (*Amer. Med. Times*, Aug., 1860). Cases of this kind are characterized by the fact that, while severe constitutional symptoms, as high fever, prostration, and great danger to life exist, there are but few local phenomena, either in the fauces or elsewhere; in fact the danger from the disease is often in inverse proportion to the extent of the membrane. Such cases can

only be explained by the hypothesis that the poison, without obtaining local footholds in the pharynx, nares, or larynx, and without giving rise to local phenomena, is directly taken up by the lungs and at once carried into the general circulation. It may affect several organs, the heart, the nerves, the blood, and the glands. Wunderlich reports two cases of Hodgkin's disease, pseudo-leukæmia, which developed during diphtheria. Bouchut and Dubri-say believe to have found a leukocythæmic condition in diphtheria. The rapid decrease of red blood-cells, and a moderate increase of leukocytes was demonstrated by them, but the disproportion was not such as to necessitate the diagnosis of leukocythæmia.

After all that has been thus far remarked concerning the localization of the deposits, and the possible differences in the symptoms, we can readily see that the course and mortality of diphtheria have a wide scope.

Most cases of diphtheria of the pharynx and of the tonsils have a favorable termination, yet a positive prognosis can in no case be given with certainty. Still even in malignant epidemics the mortality is not very great, for even though there be a large number of severe cases in any one epidemic, yet it is greatly overbalanced by the number of moderately severe and mild ones. True, not a few cases end fatally in several days, owing to the high fever, or to septic absorption, or nephritis, or croup, but the majority of cases end in recovery in one or two weeks. Yet diphtheria does not always take so regular a course; not infrequently, after the pulse has become stronger, the appetite improved, the pharynx cleared, and the patient was apparently on the high-road to recovery, another attack occurs accompanied by fever, as before, and a rapid membrane formation. Occasionally two or three such relapses may occur in the course of three, four, or five weeks; not to speak of the fact that those who have once suffered from diphtheria are more susceptible to the action

of the diphtheritic poison than those who have never experienced an attack of the disease.

THE NERVOUS SYSTEM

is in manifold ways implicated in the diphtheritic process.

The direct and rapid introduction into the blood of a foreign substance has, in almost all cases, as its earliest symptom, fever. Still we may safely assert that, with the exception of certain individual idiosyncrasies, high fever does not always result from the rapid absorption of a large amount of poisonous material. For many a septic fever runs through its entire course without any noteworthy elevation of temperature. Naturally the quantity of the poison absorbed depends on its source as well as on the individual susceptibility, and we have not in all cases to deal with a large amount of absorbed material. It has seemed to me that a series of cases depended on a gradual and protracted absorption of the poison into the blood, during a period of time, in which either elimination would take place or the septic material would exhaust itself by the chemical changes it will always undergo in a certain time.*

* Of a peculiar interest are the following facts, which prove that the substances credited with being the very strongest poisons, not only exhaust their own powers, but turn into the very opposite of what they were before. Salkowsky, in order to increase the rapidity of development in bacteria colonies, added some ascitic liquor which had been kept three years and had gone through all stages of putrefaction. Instead, however, of increasing, it *stopped* the development of bacteria (Berl. klin. Wochensch., No. 12, 1875). E. Baumann found phenol—one of the strongest anti-bacteric poisons known—as the final termination of the process of putrefaction (Zeitschr. f. physiol. Chemie, I., p. 60). In addition, in the same way Brieger discovered another antiseptic agent, scatol (indol having been found previously), and E. and H. Salkowsky hydrocinnamonic and phenylacetic acids (Wernich, in Berl. kl. Wochensch., No. 5, 1880). Thus, after a while, every one of the infectious poisons may turn into its own antidote. Who knows but that the rapidity with which many who suffered from severe infectious fevers (mainly typhoid) regain their former strength, and more than that, in a short time, depends on just such changes in the very poisons which in the beginning had the tendency to destroy life?

Thus in the course of time various localities and various centres become affected, and thus produce either cases of gradual or sudden collapse, or of gradual or sudden paralysis. The first case of gradual loss of strength and final death, in regard to which at that time I had no explanation to offer, was published by me in 1860.

The history was that of a strong, healthy boy of four years, who complained of painful deglutition, to a slight degree, and seemed languid and sleepy. At that time (it was in the autumn of 1857) no extensive epidemic had occurred in the city. The child did not appear seriously ill; there were but slight changes in the throat, a moderate swelling of the tonsils, no unusual degree of local hyperæmia, and but little deposit of membrane on each tonsil. The pulse was weak, ninety beats per minute, skin moderately warm, rather sallow and dry, extremities not cold. The child was prostrated, depressed, took but little food when it was given him, did not ask for any, and it appeared to afford him no satisfaction; local pain was not present, with the exception of a slight discomfort when pressure was made over the tonsils; bowels were constipated. The pupils responded; the boy replied in a sensible manner to the questions put to him, but rather slowly and indolently; there were no other brain symptoms present. Thus with the exception of the diphtheria of the fauces, having at that time seen no similar form of general diphtheritic infection, I could make no other diagnosis. By the second or third day, there were no changes other than that the boy seemed to grow more indifferent, depressed, and melancholy, cared nothing for the efforts made in his behalf, and expressed neither wish nor repugnance. Meanwhile the throat symptoms became somewhat more marked, the membrane covered about two-thirds of the tonsils, did not extend downward, and did not implicate the respiratory apparatus further than to cause an occasional sigh to be heard. By the morning of the fourth day, no marked change had occurred, with the exception of an increased

debility. Thus the condition became gradually worse, although the child took some nourishment, occasionally uttered a few words, replied to questions, and apparently was in the full possession of his consciousness. The temperature sank, the child became weaker, and died in the afternoon. With the exception of a more frequent recurrence of the sighs, and more rapid respiration from time to time, no change preceded dissolution. The autopsy yielded only negative results, the viscera did not appear abnormal, and were in general bloodless. The blood-vessels contained but little blood, thin and dark-colored; with the exception of the throat, no diphtheritic membrane could be discovered.

Of course, the results of this autopsy are very unsatisfactory. Microscopical examination of the tissues was not resorted to.

Unfortunately, the above case happened nearly a quarter of a century ago, that is to say, before Zenker and his followers had discovered and described the parenchymatous inflammation and the granular degeneration brought about in most of the tissues by febrile and infectious diseases. The only lesion of value in my case was the dark color and feeble consistency of the blood, which has since been found, together with extravasations, friability, and granular degeneration of the tissues, and now and then collections of cells and granules between the fibres in cases of septic poisoning and sudden death. In such cases, Hiller and Mosler lay marked stress upon the degeneration of the muscular tissue of the heart, others upon heart-clots, depending either upon insufficient contraction of the heart, or upon thrombi which have formed in remote veins, owing to a sluggishness of the general circulation; occasionally, too, upon thrombi arising in the small veins of the neck during the efforts at and interruptions of respiration by an attack of croup. Others, still, hold to the view that the sudden death is caused by an interrupted innervation of the heart. Now, either the pneumogastric

or the cardiac filaments of the sympathetic may be affected, and the symptoms will vary accordingly. Paralysis of the former will accelerate the pulse, degeneration of the sympathetic nerves will diminish its frequency, yet death may ensue in either case. The same variations, as far as heart and artery-beats are concerned, according to the locality of the degeneration, are seen in the ordinary forms of fatty degeneration of the heart in adults. The explanation of a large number of such cases of sudden death has to be sought for similarly to what we do for the symptoms pertaining to actual diphtheritic paralysis, cases of which have been reported now and then in the literature of the past, but which has been brought more vividly before the minds of the medical profession through the influence of Maingault's monographs on that subject (1854 and 1859). The differences between diphtheritic paralysis and some of the cases of sudden death are certainly those of degree and not of kind.

It was long believed that the poison exerted a local influence on individual nerves or groups of nerves. Oertel takes it for granted that diphtheritic paralysis is a progressive peripheral paralysis. At the same time, he holds that every case has its origin in the fauces, but confesses that diphtheritic paralysis is of a very peculiar nature, as it invades the nerves of a locality that has already, or appears to have, been restored to its normal condition, and occasionally attacks distant nerve territories either suddenly or gradually. Now, the hypothesis that diphtheritic paralysis originates under all circumstances in the arches of the soft palate is decidedly erroneous. A large number of observers report cases in which other parts of the body, for instance, the muscles of accommodation of the eye, were the first to become affected. I have observed cases, as have likewise Buckey and Bartels, in which the arches of the palate were entirely spared, and the cases published by Scheby-Buch clearly demonstrate that a large number of cases run their course accompanied

by a paralysis of the accommodation muscles of the eye without a similar affection of the soft palate. A case of uncomplicated paresis of the apparatus of accommodation is reported by Demoux (Gaz. Hôp., No. 108, 1877). Magne (des paral. diphthér., Paris, 1878) claims the sense of taste to be affected with paresis before other paralytic symptoms make their appearance. Equally erroneous is the view of Oertel, that the severity of the paralysis depends on the severity of the original sickness, for not infrequently it is just those cases in which neither fever nor local phenomena were of marked intensity which are followed by paralyses. I also consider the statement that it is characteristic for the paralytic phenomena, beginning with those of the soft palate, to follow a certain order, in diphtheria, as unjustifiable as the other hypothesis. This, no doubt, frequently occurs, but nowhere have I met with so many exceptions to the rule as here. I have abundant evidence that there are many cases in which *precisely the contrary holds true*, that is to say, *that it is characteristic of diphtheritic paralyses that they follow no certain course, passing by certain parts of the body and attacking others*.

It may be positively asserted that diphtheritic paralysis does not in every case depend on one and the same cause. Many cases may be identical with those arising from typhoid or from any severe infectious fever—variola, dysentery, etc. In some cases, paralysis has certainly at once crept forth, as it were, from the part first affected; in others, paralysis has followed, or gone hand and hand with, a fatty or granular degeneration of the muscular fibres, or with capillary hemorrhages and amyloid degeneration. In isolated cases, paralysis is certainly of central origin; thus Buhl discovered apoplexies in the spinal ganglia and in the gray matter of the spinal cord. In other cases, it may be attributed to the facility with which nervous disturbances and hemorrhages arise in all cases of hyperæmia, particularly where the walls of the blood-vessels are but tardily restored to their normal con-

dition, and where, in a gradual but slow convalescence, the heart is stimulated to overwork. I was formerly inclined to explain most cases of diphtheritic paralysis on the assumption that, precisely during the period of convalescence, when the patient presumes to put his strength to the test, the insufficient nutrition of the coats of the blood-vessels lead to hemorrhages, or to serous effusions. Many cases I do not hesitate to attribute to anomalies of nutrition of nervous tissue caused by the poison, which, *unaccompanied by high fever* or an extensive membrane-formation in throat and nose, is *slowly* absorbed by the lungs. Even Trousseau and Maingault had attributed diphtheritic paralysis to general infection. This view is all the more appropriate in those cases in which the process begins insiduously, and gives rise to no very urgent symptoms. The higher the fever, the greater may be the aggregation of infectious matters in the blood, but in all probability the more rapid and thorough will be the process of elimination. This would best explain the fact that diphtheritic paralysis frequently *s pares the severest cases, while visiting the apparently mildest. Only when the local manifestations of diphtheria in the throat are very marked, can we presume with some degree of certainty that the latter, too, constitute the starting-point of the paralysis.* In such cases the local paralysis, particularly when it begins early, depends first on the œdematous infiltration of the whole tissue, and further on a direct implication of the nerves or on their compression by the inflammatory products. I have remarked that in these cases, as a rule, *general* paralysis does *not* occur. On the other hand, in mild cases, paralyzes of the superior and inferior laryngeal nerves, and of the spinal nerves supplying head and extremities, occur either separately or in manifold combinations. The fact that the paralyzed nerves do not necessarily belong to the same locality, would indicate that the circulation influences the distribution of the poison. I look upon this explanation as simpler and more rational than the one

which supposes bacteria, starting at a certain locality, to bore their way through the tissues and the coats of the blood-vessels, until they are finally deposited in the muscles and nerves. Through the blood, the alkalinity of which they cannot endure, they could not be circulated into distant parts. It is true, however, that paralysis of the soft palate occurs very frequently. When not present during the first few days of sickness, as a result of local deposits or of inflammatory swelling, it appears, as a rule, in the course of the second week, sometimes, however, only after several weeks have elapsed, and even during convalescence. It occurs at times alone, at other times together with a paresis or paralysis of the constrictors of the pharynx. According to the extent of the paralysis, deglutition becomes difficult, fluids, when swallowed, regurgitate through the nose, and speech is interfered with, or these conditions do not manifest themselves very markedly. Next in frequency to paralysis of the soft palate comes *paralysis of the power of accommodation of the ocular muscles*. Eulenburg, after a large number of paralysees of single muscles of the eye had been reported and described, laid marked stress on the paralysis of the ciliary nerves, with consequent paralysis of accommodation, and asserts that this affection is apt to be symmetrical. Paralysees of the rectus internus and externus occur less symmetrically, but more frequently alternate.

Scheby-Buch gives a résumé of all the material relating to paralysis of the power of accommodation of the eye that had collected in the clinique at Kiel from 1862 to 1869. Of 38 cases, 24 had resulted from diphtheria. Of these, 20 had been diphtheritic affections of the throat, 3 of wounds, 1 each of vagina and skin. In all of those cases of paralysis of accommodation after diphtheria, mydriasis was absent, with one exception. In the other cases it was also but slightly marked. Diminished refraction occurred invariably, disappearing with the restoration to health. Occasionally a lessening of the acuteness of vision was

noticed, which disappeared together with the paralysis of accommodation. The diagnosis could often be made by the failure to observe any change of position of the lens during changes of accommodation. Paralysis of accommodation was complicated with paralysis of the soft palate in ten cases, among which there was one case of paralysis of the lower extremities in addition; in nine cases it was uncomplicated, and Scheby-Buch therefore justly infers that paralysis of the soft palate is not a *conditio sine qua non* of the occurrence of paralysis of accommodation. He refers, in this connection, to Dixon, Rook, Pagenstecher, and Roger, who have likewise seen cases of uncomplicated paralysis of accommodation. Still the latter may occur from several days (Weber, 18) to six weeks after the appearance of paralysis of the soft palate.

Next in frequency come *paralyses of the lower and upper extremities*. They rarely arise suddenly. As a rule, they involve a series of muscles at the same time, improving in the same order as the individual muscles became affected. After the paralysis has lasted some time, the circulation begins to suffer. The extremities now and then become bluish, cool, emaciated, nay even complete fatty degeneration and atrophy have been observed; such manifestations as the latter are, however, exceptional. Not infrequently the muscles of the neck are affected; the head cannot then be held erect, or this is accomplished with difficulty.

One of the cases of diphtheritic paralysis reported by F. Pennavaria (Il Morg., Aug., Sep., 1877) is of great interest, inasmuch as he emphasizes the paralysis in the fingers, the occurrence of which has been denied by several authors. The diphtheria of the patient (boy of sixteen years) was cured in twenty-four days, but paralysis of the pharyngeal muscles and consecutive dysphagia followed. Four days afterward, there were disorders of accommodation, which lasted a fortnight. After this, paralysis of both *lower* extremities, and paresis of several fingers. Complete recovery resulted

from eight faradizations. His other case was that of a child of three years, whose pharyngeal diphtheria lasted sixteen days. Paralysis of the soft palate and pharyngeal muscles followed; a month afterward universal anasarca. When it disappeared rather suddenly, œdema of the glottis set in. This also passed by, and deafness and paraplegia had their turn. When the boy was examined three years after, he was well, with the exception of permanently hard hearing.

The *bladder* is rarely affected; likewise the *intestinal sphincters*. Among the most dangerous cases are those in which the *respiratory muscles* are involved in the paralysis. If paralysis of the pharyngeal muscles be in so far dangerous, and even fatal, that food may enter the larynx and the remaining air-passages and give rise to direct suffocation or to a secondary pneumonia, then paralysis of the respiratory muscles must be looked upon as far more dangerous to life. I recollect one such case which followed a paralysis of the arches of the palate and another independent of any preceding paralysis of the palate. Though the progress of diphtheritic paralysis in general be mild, and the prognosis in general favorable, these cases are apt to terminate fatally. There is more danger of such a result than of a permanent paralysis of nerves and muscles. True, there have been reports of permanent paralyses; I have never seen any, however. I have even witnessed complete restoration to health after atrophy had lasted for months.

Thus far I have spoken only of motor paralyses. They are by all means the more frequent, but *sensitive paralyses* may occur in the same manner. Frerichs and Gerhardt have called attention to pure anæsthesias. I have myself observed a pure anæsthesia of the upper portion of the trunk. The association of motor and sensory paralysis may also be observed, giving the appearance of *locomotor ataxia*. A case of ataxia following diphtheria has been described by Rumpf (Deutsch. Arch. f. klin. Med., 1877, XX., p. 120); another by R. Schulz (1879, XXIII., p. 360).

A fortnight after recovery from diphtheria was completed, the patient complained of double vision, deficient vision at long or short distances, and dizziness; soon afterwards nasal voice, difficulty in swallowing, liquids returning through the nares, increasing debility in both arms and legs to such an extent as to render his gait unsteady; formication in the limbs, and anæsthesia of the soles of his feet. When the author first saw him, the muscular power of the extremities, particularly the right, was diminished, but sensibility, with the exception of formication, intact. Pressure upon the trunks of the nerves of the extremities, and upon the ganglion supremum of the sympathetic was very painful. Gait unsteady, atactic, more with closed than open eyes. Patellar reflex entirely gone. Faradic examination revealed the irritability of the radial nerves but little reduced, if at all; nor is the galvanic examination any more conclusive. No degeneration of the muscular tissue, to judge from electrical or galvanic tests. No cerebral disorders whatsoever, for the dizziness was owing to the paretic condition of the rectus internus muscle.

The treatment consisted of galvanization through mastoid processes with six elements daily at first, every other day afterwards; also positive pole on neck, negative on eyelids; ascending current over cerebral column with sixteen to eighteen elements; galvanization of extremities sufficient to yield muscular contraction, of soft palate with six elements. The paresis of accommodation was improved after six sessions, removed after twelve, that of rectus internus and palate cured after twenty. Treatment having then been interrupted for a fortnight until the sensitiveness of the nerves of the extremities passed by, seven more applications of the galvanic current removed both paresis and ataxy of the extremities, so that the patient returned to work. At that time, however, the tendon reflex had not returned at all.*

* Deutsch. Arch. f. klin. Med., 1879, XXIII., p. 360.

The results of electrical applications have frequently been noticed, but the reports differ as to their nature. The effect of the continuous current was found normal by Caspari and Rumpf, greatly diminished by Leube; Krafft-Ebing found the action of the interrupted current gone, the galvanic normal; Rosenthal and Jaffray, the former reduced, the latter increased. In my hands, the response to electricity was not the same in all cases. Very frequently in the beginning the response to the induction current was normal, sometimes deficient; to the galvanic current exaggerated; after some time the power of both to excite a reaction diminished. But when we reflect on the numerous causes that may underlie the diphtheritic paralysis, and that we have not in every case to deal with one and the same process, it will become apparent that the reaction to the galvanic current cannot be the same in peripheral, spinal, or cerebral paralysis. The first are certainly more frequent. It is possible, however, that in future the very observation of the action of the current may be rendered useful for obtaining a local diagnosis.

There are but few autopsies of cases which died of, or during, diphtheritic paralysis. There was considerable thickening of the spinal nerves at the junction of the posterior and anterior roots, with hemorrhages. The superficial connective tissue in these places exhibited a diphtheritic exudation (Buhl). There was in the sheath of the nerves in the cerebral and spinal meninges and in the gray substance of the cord voluminous nuclear infiltration, in one case there were extensive hemorrhages in the spinal meninges, with nuclear proliferation in the gray substance of the cord (Oertel). Also disseminated meningitis with perineuritis of the neighboring roots, characterized by infiltration of nuclei between the nerve fibrillæ (Pierret). Also degeneration of the palatine nerves and fatty disintegration of the palatine muscles (Charcot and Vulpian). Dejerine, in five autopsies, records an atrophy of the anterior roots secondary to a myelitic degeneration

of the ganglia of the anterior horns. In two cases he reports finding the same changes in the intramuscular nerves, viz., liquefaction of myelin and loss of axis cylinders.

Thus Buhl, Charcot, Vulpian, and Dejerine are unanimous about an affection of the peripheric nerves and muscles. Oertel and Dejerine believe in a disease of the spinal cord. It is true that a disease of the gray substance would fully explain the symptoms mainly of the bad cases, but what we know of poliomyelitis anterior, with which this affection would be identical, precludes the idea of that rapid, and almost certain complete recovery. Therefore, in most cases, diphtheritic paralysis consists of a trophic affection of the motory system, almost always peripherically in nerves and muscles, seldom, if ever, in the centre. This affection must be compared, in most bearings, with the degenerative processes taking place in the muscular tissue after typhoid fever, in the renal epithelium after infectious diseases, both of which give rise to serious results, with mostly a favorable termination.*

SUMMARY.

The first invasion of pharyngeal diphtheria resembles sometimes very much that of a catarrhal pharyngitis. The latter is general. Local hyperæmia points to either trauma or diphtheria.

Three forms of diphtheria are found in the fauces: the "croupous," the "diphtheritic," and the "necrotic."

Glandular swelling about the neck is not always very marked. The above three species of diphtheria may each be found in mild or severe attacks. The last is apt to become septic and fatal. Nasal diphtheria is either the continuation of the process from the soft palate, or primary. It is complicated with and characterized by rapid swelling of the deep-seated facial glands in most cases.

* See Dr. Fritz, *Charité-Annalen*, V., 1880, p. 255.

A chronic catarrh of pharynx, nares, and larynx is some times observed after the acute attack.

Diphtheria of the conjunctiva terminates often in destruction of the sclerotic and prolapse of the iris. It is frequently the only symptom of diphtheria, and purely local.

The ear is affected either through the Eustachian tube, or in and from the external auditory canal.

Diphtheria of the epiglottis is rarely found extensively on the upper surface, more on the lower, and sometimes in more or less isolated spots on the free margin. When complicated with but slight laryngeal affection, the croupy symptoms are but mild. They are apt to be of long duration.

Local œdematous infiltration of the upper posterior portion of the larynx interferes with inspiration more than with expiration; membranous deposits in the larynx with both, and result in the worst forms of "membranous croup." Tracheal diphtheria is mostly the result of descending laryngeal membrane. But there are cases of primary tracheal diphtheria which, when ascending, result in speedy suffocation.

The lungs may be the seat of either broncho-pneumonia from several causes, or fibrinous pneumonia. The diagnosis is very difficult, both auscultation and percussion yielding but doubtful results, unless there is a sudden increase of fever and of respiratory movements. Blood entering the lungs during tracheotomy may result in broncho-pneumonia.

Diphtheria of the mouth is not frequently primary, mostly secondary, and the deposits are first seen on sore surfaces. The same is true in regard to the œsophagus. Its upper portion is often affected in cases of pharyngo-laryngeal diphtheria. Solid fibrinous deposits are met with in typhoid fever, variola, and other infectious diseases.

The intestine is affected with diphtheria (beside the

dysenteric process) in its upper and lower portions. In animals very extensive intestinal diphtheria has been observed.

Recent wounds are liable to be affected with every one of the three forms of diphtheria within a day, or later, after an operation. Local or general cutaneous erythema is sometimes found. Complications of diphtheria with erysipelas are not uncommon, and dangerous. The bladder, in cystitis, or after operative procedures, vagina when eroded, prepuce when operated upon, and placenta are the seats of diphtheritic deposits.

Albuminuria is frequent, is mostly not dangerous, accompanies sometimes a rapid process of elimination of the poison, occurs often at an early period of the disease, does not depend on, nor does it increase, the fever, seldom lasts over a week, but is sometimes one of the symptoms of diffuse acute nephritis of a very grave character.

The heart may suffer from defective innervation, granular degeneration, thrombosis, or endocarditis. The blood may be of a dark color, but is not leukocythæmic. Pseudo-leukæmia, however, has been observed during diphtheria.

Thus the course of diphtheria is very various, prognosis doubtful, relapses are frequent, the temperature of the blood is not pathognomonic, the amount and rapidity of absorption and elimination changing.

The nervous system suffers often; sometimes in the very beginning of the disease collapse is developing, and may lead speedily to a fatal termination.

Diphtheritic paralysis is considered peripheral by some, central by others. It does not always commence in the soft palate; the latter has been known not to participate in the paralysis at all. The muscles of accommodation are frequently affected, also the extremities, in some instances sensory nerves, sometimes the respiratory apparatus with dangerous results. Paralysis occurs mostly during convalescence from diphtheria, exhibits no regularity in the succession of the parts affected, does not injure

the sphincters, and is in most cases amenable to treatment. While, in the majority of cases, the disease appears to strike the trophic fibres of the motory nerves, the action of the electric and galvanic currents is very variable. In but a few cases ataxy has been observed.

CHAPTER VI.

ANATOMICAL APPEARANCES.

The membrane, or the granular infiltration are characteristic of diphtheria. The statement that the former occurs only when atmospheric air can gain access thereto, as A. D'Espine and C. Picot still hold (*Man. prat. des mal. de l'enfance*, 1877, p. 81), is plainly contradicted by its appearance on the mucous membrane of the lower intestines. The condition of the membrane is not unalterable, any more than the clinical symptoms of the disease, for according to different circumstances, epithelium, mucus, blood, and vegetable parasites are added thereto. The membrane can either be lifted from the mucous surface on which it lies, or is imbedded into and underneath it. In the first instance, it consists to a great part of fibrin, the result either of epithelial changes, or derived directly from the exuded blood-serum. E. Wagner considers epithelial changes the principal source. He makes no anatomical distinction between croup and diphtheria. The pavement epithelium becomes altered in a peculiar manner. It becomes turbid, larger, dentated, and dissolves into a network; it is at first uninhabited, but serves later as the vehicle of newly formed cells. Rapid metamorphosis of epithelium, and an equally rapid new formation, are the conditions of a speedy and extensive membrane formation. The same changes occur in granulation tissue, the granulating surface of an ulceration in the mouth exhibiting the same process as the mucous membrane. In addition to the formation of membrane on the surface, there also occurs a considerable infiltration of the mucous membrane with pus-cells and granules; be-

sides, the cellular tissue is studded with granules, its decomposition resulting in fine granular deposits in the tissues and necrotic destruction, which is looked upon by Virchow as the most important element in severe forms of diphtheria.

These several conditions may occur independently, associated, or in succession.

Classen, with E. Wagner, considers an alteration in the superficial layer of epithelial cells characteristic of diphtheria. They become amorphous and the entire mass indistinctly reticulated; this change becomes the more decided the longer the membrane existed. Solution of caustic potash demonstrates peculiarly altered cells therein, the contents of which are finely granular. These dark granules, which strongly refract light, are "perhaps identical" with the monads of diphtheritic blood described by Hüter and Tommasi. "If so, the disease might be considered as beginning with an enormous proliferation of these minute bodies in the epithelial coating, followed by a local fatty degeneration of the mucous membrane." These bodies would, presumably, circulate in the blood.

Wagner's doctrines are assailed by Boldygreu, who attaches importance to the parallel condition of the layers, and to the appearance of peculiar concentrically-lamellated spheroidal bodies endowed with a dark and granular centre. These have already been declared to be, in all probability, drops of exuded mucous secretion which then give rise to a peculiar configuration of the exudation. It is said that ciliated epithelium is never seen on a mucous membrane affected with croup; but numerous papillary elevations consisting of granulation tissue and pus corpuscles, each covered with a sheath, present themselves; furthermore it is asserted that hemorrhages do not occur in croup as in diphtheria; and whereas the latter is characterized by an infiltration of small cells, a finely granular reticulated product occurs in the former. According to Boldygreu, the membrane consists of successive coagula-

tions of a fibrinous fluid which exuded from the diseased surface.

Steudener opposes the views of Wagner, remarking that he has never seen the early stages of a metamorphosis of cylindrical epithelium such as Wagner describes in connection with the pavement epithelium of the pharynx. He does not believe in the probability of an exclusively endogenous origin of the cellular elements of croup membrane; in fact, he doubts the occurrence of an endogenous formation of pus globules in epithelium. Croupous membrane, according to him, is formed by the migration of numerous white blood-globules through the walls of the vessels in the mucous membrane, and by a direct formation of fibrin from the transuded plasma. In addition to this, the mucous membrane is stripped of its epithelium (except at the mouths of the acinous glands), and infiltrated with migrating cells. The latter are most numerous immediately under the surface, with the exception of the thin, pale, superficial border of the basement membrane. The vessels and ducts of the glands thereby become compressed, and the glands distended. Migrating cells are less found in the deeper tissues, where elastic fibres are in larger amount than superficially; an extensive infiltration exists in the submucous tissues, especially about the glands and even in the connective tissue surrounding the cartilaginous rings of the trachea. Fresh croupous membrane consists of a delicate network, of homogeneous structure and shining appearance, in which numerous cells and the epithelium of the various layers of the trachea are imbedded. There are, moreover, places where the cells are few in number and here and there, too, membranes consisting (according to Rindfleisch) of round, densely agglomerated cells. In old membranes, the cells are destroyed by granular degeneration and general maceration. Tenacious mucus with pus-cells and detritus are then found.

The doctrine which claims that the diphtheritic process

is caused, excited, or aided by bacteria, has been sustained in the most varied manner by Hallier, Laycock, Wade, Hüter, Oertel, Klebs, Eberth, and many others. I have discussed this question in an earlier part of this treatise. Karsten (*Wien. med. Woch.*, 1873, 39) looks upon bacteria as a pathological cellular formation, analogous to pus and yeast cells; and asserts that they originate within the cells and are not introduced from without.

Attempts at producing artificial diphtheritic membrane were made long ago. Already in 1826 Bretonneau, by the introduction of tincture of cantharides and olive oil into the trachea, succeeded in producing a "dense, elastic, reed-like membranous concretion." Delafond called "croup" into existence by the use of ammonia, oxygen, chlorine, corrosive sublimate, arsenic, and sulphuric acid. On the other hand, H. Mayer asserts that it is impossible, by means of ammonia, to produce a croup in the windpipes of animals which in the slightest degree resembles that occurring in human beings. Trendelenburg, after producing membranes in the trachea by the use of a solution of corrosive sublimate (1:120) succeeded in hardening the entire mass with bichromate of potash, which it was impossible to do with the most tenacious mucus.

Rey observed croup in horses that inhaled smoke in a burning stable (*Journ. de méd. vét. de Lyon*, 1850, p. 249). In the collection of the veterinary school of Zurich, there is a croup membrane from a heifer which had been exposed to a fire; at Munich, one from the trachea of a horse, produced by forcibly injecting medicines into the nose. Hahn made an observation on cows, W. Ammon on horses, of long croup membranes, after the animals had been exposed to smoke and fire, and Oertel constantly insists on there being "no actual difference between croup as it ordinarily occurs, and that excited in the windpipe of a rabbit by means of ammonia. The color and texture, the physical, chemical, and histological characteristics are identical." In regard to this question of identity or non-

identity, the very last writer on the subject, C. Gerhardt (Lehrb. d. Kinderkrankh., IV. ed., 1880, p. 297) expresses himself in the following manner: "The difference between laryngeal diphtheria and croup, after having been so much emphasized, cannot be sustained any longer. It was claimed by anatomists that diphtheritic membranes were imbedded into and closely attached to the mucous membrane, the croupous, however, lay closely upon it and were easily separated from it, without leaving any ulceration behind. If that were so, we should always see diphtheria of the larynx changing into croup of the trachea. Not more successful is the attempt at an etiological distinction of the two forms, by assuming that diphtheria is epidemic and croup sporadic. I have myself observed and described a sporadic case which proved contagious. Besides, the schizomycetæ connected with diphtheria exhibit great differences when we come to examine the descriptions and drawings."* On the other hand, C. Weigert (Virch. Arch., Vol. 70), who experimented on artificial croup produced by the application of caustic ammonia, has his own theory on the origin of the pseudo-membrane. It does not originate in the epithelium, for this may be absent and still the membrane will develop. Thus it originates in parts belonging to the cellular tissue. He deems both Oertel's assertion, that the croup membrane results from the conglutination of large protoplasmic cells, and the assumption that the glands could yield a solidifying secretion, incor-

* To what excesses the bacteriomania can guide the mind is, in contrast to the above, illustrated by A. Ott, in Prag. Med. Woch., No. 11, 1880. Five days, eight days, and five weeks respectively, after three cases of pharyngeal diphtheria in two adults and one boy, he observed fever and pain about the chest. This pain did not follow the course of the nerves, and increased on pressure. Some days afterwards pneumonia was diagnosed. One of the cases terminated fatally. The reader is coolly requested to take both pain and pneumonia as the result of parasitic infection depending on the preceding diphtheria, and is given his choice between either of two theories, viz., that the parasites migrated from the painful muscles (?) to the lungs, or that they invaded both muscles and lungs through the lymph circulation.

rect. He looks upon the deposits as analogous to those on serous membranes. Every inflammation yields an exudation which may coagulate when the coagulating ferment is added. This latter is probably produced by the white blood-cells when in disintegration. But he does not say why it is that there is no such coagulation in suppurative processes, where the leukocytes are more numerous. And does the experiment with ammonia, which necessarily destroys the surface epithelium, solve the problem of the origin of membranes when the epithelium is not forcibly removed or destroyed?

Weigert also believes himself justified in establishing pathological differences of croup, pseudo-diphtheria, and diphtheria. A croupous inflammation means destruction of epithelium, which gives rise to a fibrinous exudation upon the surface, while the cellular tissue remains intact. The only difference between it and the pseudo-diphtheritic inflammation is looked for in the larger number of emigrated white blood-cells. The superficial deposit consists, to a great part, of them and the fibrinous exudation. When there are but few leukocytes, the deposit is a network of fibrillæ ("croup"). When there are many, the masses are more solid and voluminous ("pseudo-diphtheritis"). When, however, the tissue is changed into a hard substance, resembling coagulated fibrin; when the exudation does not exist on the surface, but takes place into the mucous membrane, the process is "diphtheria." Finally, J. Zahn publishes contributions to the pathological histology of diphtheria which are interesting and important. Careful examinations of various diphtheritic membranes, mostly taken from the living, induce him to establish three varieties, viz.: 1st, such as result from a peculiar degeneration of pavement-epithelium; 2d, such as originate in the solidification of a muco-fibrinous, and 3d, of a fibrino-purulent exudation. Each of these varieties may contain colonies of micrococci (gliococci, megalococci, and bacteria), but these organisms are neither essential nor are

they constantly met with. In the adjoining tissues, either normal or infiltrated with round cells, particularly in the uvula, the parasites were not found. Thus the author prefers not to venture upon a decided answer to the questions of their essentiality.

Klebs, as may be expected from the position he always maintained in the diphtheria question, is less hesitating (*Beitr. z. pathol. Anat.*, 1878, p. 11). He admits that the membrane may be, or is, the same histologically in all cases, no matter how much the clinical symptoms may differ, but adds that, in the diphtheritic membrane, there is a development of bacteria, of always the same character, and pathognomonic. The last word is a gratuitous appendage which is not proven, as we know. The constant repetition of the same statements does not render them any the sounder.

However, the diphtheritic process does not merely consist of the changes in the pharynx and air-passages. Its fatal cases have afforded marked evidence of the implication of most of the organs. Reimer's 17 cases give the following post-mortem results: the lungs were hyperæmic in 8 cases, twice the seat of pneumonia, and three times of embolic infarctions. In addition, emphysema in 12, œdema in 6, atelectasis in 7, subpleural ecchymoses in 7, pericardial ones in 4. The heart-muscle had undergone fatty degeneration in 6, and was the seat of ecchymoses of the size of a pin's head in 3. In addition to frequent hyperæmic conditions of the abdominal viscera, emboli of the liver in 3 (with capillary hemorrhages of the peritoneal covering in 1), emboli of the spleen in 5, desquamative nephritis in 7 (in 6 of which there were colonies of micrococci in the uriniferous tubules), cellular hyperplasia of the cervical and mediastinal glands in 14 (complicated in 6 with capillary hemorrhages in the glandular tissue). The *blood* was frequently normal, very often watery and dark, at times leukocythotic.

In the *heart*, particularly the right, numerous thrombi in

various stages of development were found; its muscular tissue was often in a state of fatty degeneration, or the seat of parenchymatous inflammation and hemorrhages. Bridges first called attention to the occurrence of endocarditis in diphtheria (*Med. Times and Gaz.*, II., p. 204). Diphtheritic endocarditis, which, however, occurs more frequently with rheumatism, puerperal fever, diphtheria of wounds, pyæmia, and old valvular affections than in the course of an acute diphtheria, does not consist simply of a fatty degeneration and subsequent ulceration, but is a genuine diphtheritic process (Virchow) affecting the mitral valve more frequently than the tricuspid or pulmonary valves. It begins with hyperæmia and the exudation of plasma in the cellular elements, so that they appear larger and darker. The granulations which form are frail and easily destroyed, so that ulcers form on which fibrin is deposited, and whence it is conveyed as emboli into the "terminal arteries" (Cohnheim) of the spleen, nerves, brain, and eye. Infarctions may also occur in the valveless veins of these organs, giving rise rather to small multiple abscesses than to large purulent collections. Suppuration but rarely takes place in the heart; the granular mass found there resists the action of ether and alcohol (parasitic? caseinous?), and spreads throughout the cardiac parenchyma, so that perforation of the septum, and of the right auricle and aorta have been observed.

Bouchut and Labadie-Lagrave, out of fifteen cases of diphtheria, met with a plastic endocarditis in fourteen, which became the source of emboli. Thus there were infarctions of the lungs, at times in their centre colorless, at other times in a state of purulent degeneration; superficial thrombi of the small veins of the heart, subcutaneous connective tissue, pia mater, brain and liver. In addition moderate leukocytosis.

THE LUNGS

exhibit, post-mortem, all sorts of inflammatory and con-

gestive conditions and hemorrhages, as: œdema, catarrh, broncho-pneumonia, atelectasis, emphysema, ecchymoses, and large infarctions.

THE SPLEEN

(and occasionally the *liver*) is frequently large, congested, and friable, and studded with infarctions to a greater or less extent.

THE KIDNEYS

are either simply congested, or the seat of nephritis or infarctions. The same forms of inflammation which accompany scarlatina, to wit, the desquamative and the diffuse, are here observed. The diffuse form is not of so frequent occurrence as in scarlatina, but sometimes extensive and dangerous.

THE MUSCLES

occasionally exhibit ecchymoses, and are at times the seat of parenchymatous inflammation, gray degeneration, and atrophy.

THE LYMPHATIC GLANDS

are frequently inflamed and swollen, either hard or doughy, œdematous or congested. Large abscesses rarely appear therein. It is more especially the gland tissue, and less the connective tissue of the glands, which takes part in the pathological process. The periglandular tissue very soon becomes involved, however. Necrotic foci have been described by Bizzozero. In relation to this and other matters, but lightly touched upon in this place, I refer to other parts of this work.

In regard to the nature of the glandular and periglandular swelling, F. Balzer and Ch. Talamon investigated the condition of the submaxillary and parotid glands. The main changes were found in the epithelium of the excretory ducts and acini; 2d, in the connective tissue, and

mainly in the pericanalicular connective tissue; 3d, in the blood-vessels which are often congested; 4th, in the lymph vessels which are dilated and filled with cells. In consequence of all this, the glands swell and become of a yellowish color. For some time, isolated swellings can be felt, afterwards they may join in a common extensive tumefaction, which yields a peculiar sensation of spurious fluctuation (Revue Mens., Juill., 1878).

INTESTINAL CANAL.

According to Virchow, diphtheria of the *intestinal canal* is characterized by fibrinous deposits on the surface and in the tissues of the intestine, with subsequent granular degeneration of the tissues. According to Rajewsky, a catarrhal process invariably precedes the diphtheritic affection of the intestines. The latter begins with deposits of fibrinous exudation in the mucous membrane and on its surface. Then follows the destruction of the tissues of the mucous membrane, and their conversion into a granular mass, containing albumen, to judge from its solubility in acetic acid. This process extends, and at the same time a hyaline metamorphosis of the blood-vessels in the affected tissues takes place. As long as the tissues remained unchanged, isolated micrococci and bacteria were observed; as soon as the granular degeneration set in, the organisms appeared in colonies. In both conditions, *i. e.*, even before the tissues underwent alteration, lacteals filled with bacteria could be noticed in the submucous tissue. Rajewsky endeavored experimentally to ascertain the exact relation bacteria bear to diphtheria of the intestines; by injecting a dilute solution of ammonia, he succeeded in creating an inflammation of the mucous membrane of the intestines. If now a fluid containing micrococci was injected into the blood of the animal, the parasites are said to have circulated freely until they reached the intestinal canal, where they held fast, multiplied speedily, and at the same time the hyaline metamorphosis of the blood-vessels

was inaugurated. When no affection of the intestines pre-existed, or the animal had not been prepared as before, diphtheria of the intestines was not observed. The latter was as little apparent after the injection of the ammoniacal solution alone, nothing more than a temporary inflammation resulting. If, however, a putrid fluid was injected immediately after, death resulted in from twenty-four to thirty-six hours. Rajewsky infers as a result of these experiments that the injection of fluids containing bacteria cannot give rise to diphtheria of the intestines except after the mucous membrane of the intestinal canal has been prepared to afford a resting-place for it, by a preceding inflammation. It is thus presumed that parasites play an important part in intestinal diphtheria. The inflammatory changes of the tissues of the mucous membrane are in intimate connection with the hyaline metamorphosis of the blood-vessels.

It seems to me that here, too, the nature of diphtheria has been coolly postulated in the interests of the parasitic theory. Exudation and granular degeneration are claimed by Virchow as the characteristic phenomena of diphtheria of the intestines; exudation, granular degeneration, *and* a multitude of parasites, by the defenders of the bacterian theory, who indissolubly connect the name and nature of diphtheria with the presence of parasites. Moreover, Rajewsky reports no changes brought about by the action of the parasites alone, and this is, after all, the most important point, since the same changes have been observed where neither the influence nor even the presence of the bacteria could be verified as a factor in the process.

Indeed, nowhere has the postulation of ideas and definitions been more confusing and detrimental than in the study of the nature of diphtheria. Of late it has been with many only a question of whether bacteria are present in the morbid product in order to declare it diphtheritic; formerly, and even now, certain differences between "croup" and "diphtheria" were and are postu-

lated, and I shall, therefore, add a few final remarks here on the question of the identity of these affections.

Can pseudo-membranous croup be distinguished from laryngeal diphtheria? Ought these terms to be preserved separately? Are they different processes? Let us suppose two cases of membranous impediment in the larynx, the one with, the other without, membrane in the pharynx, the other symptoms being the same, is one "diphtheria of the larynx," and the other "croup"? Suppose again, a membranous stenosis of the larynx, to which is only later added a membrane of the pharynx, was the case originally one of "croup" which became a "diphtheria" later on? Thirdly, take two cases of laryngeal stenosis, one with symptoms of suffocation only, the other having these symptoms together with adynamia; is the latter "diphtheria" alone, the former only "croup"? In my opinion, it is just as little possible to differentiate these diseases according to the seat of the morbid product, as it is justifiable to deny the title diphtheria to membranous pharyngitis when few general symptoms, such as fever, debility, and collapse, happen to be present.

Even Monti declares croupous laryngitis to be a separate disease, independent of diphtheria, *but* considers that it may arise from a diphtheritic contagion; he still, however, treats of the disease in two distinct chapters, calling the one laryngitis crouposa, the other laryngitis diphtheritica. On the other hand, it is a well-known fact that Senator and the microscopists have declared these diseases to differ in degree, although identical in character. Fleischmann thinks that the pathological picture is obscured by the occasional combination of croupous and diphtheritic laryngitis, but that such cases are the last ones from which any conclusions should be drawn, and "it would seem as though these were the very cases employed preferably by some physicians to perpetuate the confusion" (*Oest. Jahrb. Päd.*, 1875, I.). I fear that "thou art the man," and that we can promise ourselves little from the

tendency to subdivide and subclassify nature. She does not work in narrow schedules. Still, not even last year's discussion in the Royal Medico-Chirurgical Society led the members to harmonious convictions.*

The different forms of the diphtheritic affection, as I have described them, have a particular preference for certain localities. When the entire mucous membranes of

* The conclusions arrived at by its committee, appointed to examine into the relations existing between "croup" and "diphtheria," are as follows (report presented by Mr. Andrews): 1. Membranous inflammation confined to or chiefly affecting the larynx or trachea may arise from a variety of causes, as follows: (a), from the diphtheritic contagion; (b), by means of foul water, of foul air, or other agents, such as are commonly concerned in the generation or transmission of zymotic diseases; (c), as an accompaniment of measles, scarlatina, or typhoid, independently of any ascertainable exposure to the especial diphtheritic infection; (d), it is stated, on apparently conclusive evidence, that membranous inflammation of the larynx and trachea may be produced by various accidental sources of irritation—the inhalation of hot water or steam, the contact of acids, the pressure of a foreign body in the larynx, and a cut throat. 2. There is evidence in cases which have fallen under the observation of members of the committee, that membranous affections of the larynx and trachea have shortly followed exposure to cold, but their knowledge of the individual cases is not sufficient to exclude the possible intervention or co-existence of other causes. The majority of cases of croupal symptoms directly traceable to cold appear to be of the nature of laryngeal catarrh. 3. Membranous inflammation, chiefly of the larynx and trachea, to which the name "membranous croup" would commonly be applied, may be imparted by an influence, epidemic or of other sort, which in other persons has produced pharyngeal diphtheria. 4. And, conversely, a person suffering with the membranous affection, chiefly of the air-passages, such as would commonly be termed membranous croup, may communicate to another a membranous condition, limited to the pharynx and tonsils, which will be commonly regarded as diphtheritic. It will thus be seen that, in the opinion of the committee, these two diseases are identical. It is suggested that the term "croup" be henceforth used wholly as a clinical definition, implying laryngeal obstruction, occurring with febrile symptoms in children, which may be membranous or not membranous, due to diphtheria or not so. The term "diphtheria" is the anatomical definition of a zymotic disease, which may or may not be attended with croup. It is admitted, however, that when obviously occurring from a zymotic cause or distinct infection, and primarily affecting the pharynx, constitutional depression is more marked, and albuminuria is more often and more largely present, though in both conditions some albumen in the urine is more frequently present than absent.

the mouth and of the air-passages, from the nose to the trachea, are the seat of the disease, there is an impregnation of the mucous membrane from the epithelial surface to the submucous tissue of the entire tongue, borders of the lips, and frequently of the lips and cheeks, as well as of the tonsils, the lower portion of the nasal cavities and the upper and especially the anterior portion of the larynx. The fossæ Morgagnii and the posterior aspect of the soft palate are more frequently affected in the same way than the anterior aspect. Small isolated spots are found on the tonsils and occasionally on the posterior wall of the pharynx. The so-called croupous form, that is to say, the one in which the membranes deposited may either be removed in large patches, or lie macerated in the profuse secretion of subjacent mucous glands, is found partly in the nasal cavities, on the posterior surface of the soft palate, in the trachea, and its subdivisions.

The tissues in general, and the mucous membrane in particular, have been supposed to be endowed with certain peculiarities which enabled them to markedly influence the diphtheritic process. Eberth has directed attention to the fact that extensive laryngeal diphtheria does not necessarily descend into the trachea, although he overestimates the numerical frequency of this exclusive localization. Trendelenburg infected the trachea of a rabbit with diphtheritic deposits which he had removed from the pharynx and tonsils, in the tissues of which they were deeply and firmly imbedded. The new deposits, however, did not take so deep and firm a hold on the tissues as the original ones, but adhered lightly to the mucous membrane of the trachea to which they had been transplanted.

It must not be forgotten that the character of the mucous membrane varies with the locality. Its different elements, as the epithelium, basement membrane, connective tissue mingled with elastic fibres, blood-vessels, nerves from the cerebro-spinal and sympathetic systems, and frequently with spindle cells, the papillæ and ducts

of numberless glands, all influence the pathological process going on upon the surface.

THE MUCOUS MEMBRANE OF THE MOUTH

contains a large number of elastic fibres mixed with cellular tissue and covered by a thick coat of pavement epithelium, whose uppermost layer contains flat cells, the second a larger quantity of polygonal cells, and the lowest oval ones which assume a perpendicular relation to the mucous membrane. From the mucous membrane a number of papillæ extend into the epithelium, and in this respect resemble the papillæ of the skin. Acinous muciparous glands are frequent, and most numerous in the anterior aspect of the soft palate. Lymph vessels are very numerous in the lips, tongue, uvula, soft palate, anterior and posterior pillars of the soft palate, and cheeks. The uvula contains so many that, if they be injected, its circumference is increased two or three fold. They empty into the deep facial glands to which they communicate the infection in diphtheria. The lymphatics of the tongue are in intimate connection with the upper layer of the deep cervical glands; those of the floor of the mouth and many from the tongue, with the submaxillary glands. The efferent vessels empty their contents into the superior jugular glands, in the trigonum cervicale superius, and finally into the fifteen or twenty inferior jugular (or supraclavicular) glands, which with numerous anastomoses form the jugular lymphatic plexus. The tonsils are conglomerations of an indefinite number of glandular bodies, each of which has a thick capsule which is of irregular shape, and consists of connective tissue lined by mucous membrane and pavement epithelium. The connective tissue contains a number of closed follicles, each inclosing numerous lymph-corpuscles. These follicles have been considered identical with, or analogous to, the lymphatic glands; this assumption is purely problematical, since it has not been possible, thus far, to verify the existence of

afferent or efferent ducts. The practical deduction from this is, that the tonsils have little or no connection with the lymphatic system. The number of blood-vessels in the normal tonsil is not large, and it becomes greatly diminished when that organ has been the seat of repeated chronic inflammations. It is in these very cases of chronic inflammation and enlargement of the tonsils, with considerable hypertrophy of the connective tissue, that diphtheritic attacks are most frequently observed, especially, of course, when a fresh irritation has given rise to inflammation and an œdematous enlargement.

THE MUCOUS MEMBRANE OF THE NASAL CAVITIES

is of varying degrees of thickness; it consists of connective-tissue fibres with numerous nuclei, is free from elastic fibres, but is supplied with a large number of nerves, and an abundance of blood-vessels; the Schneiderian membrane possesses in fact a larger number of blood-vessels than most of the other mucous membranes. It is, therefore, with its submucous tissue, the seat of frequent swellings and hemorrhages, as well in diseases of distant organs which give rise to venous stagnation, as from the slightest local provocation. The inner surface of the cartilaginous portion is lined with pavement epithelium; the lower region of the real nasal cavities, the so-called respiratory portion, through its whole length supplied with branches of the trigeminus, is lined with cylindrical epithelium, and contains a large number of mucous glands. The upper or so-called olfactory portion is lined with ciliated epithelium, and is supplied, according to Todd and Bowman, with long, straight tubular glands. According to Max Schultze, certain of the epithelial cells, particularly those belonging to the layer of oval, bipolar ones, are in connection with the terminal ends of the olfactory nerves. They have been designated as olfactory cells. In the last-named portion of the nasal cavities the lymphatics are but poorly developed, while in the inferior portion they are

very numerous; all their openings communicate directly with the deep facial and posterior submaxillary glands. Thus it can be readily understood why the slightest irritation, by a nasal catarrh for instance, in a child produces a temporary or permanent swelling of the glands.

THE EPIGLOTTIS

carries pavement-epithelium of 0.2 mm. in thickness on its anterior superior surface, that on its posterior surface being from 0.06 to 0.1 mm. in thickness. The superficial layer consists of spheroidal or polygonal cells, the deeper, of cylindrical cells, is arranged perpendicularly to the surface. Near the attached extremity of the epiglottis, the polygonal cells disappear, the cylindrical occupy the surface, and are furnished with cilia 0.005 mm. in thickness. Beneath these there are round and oval cells in considerable number, so that the whole epithelial coating has a thickness of 0.510 mm. Ciliated epithelium is also found on the false vocal cords and in the ventricles of the larynx. Polygonal pavement-epithelium forms the covering of the posterior surface of the pharynx, of the ary-epiglottidean folds, where the mucous membrane possesses, in addition, a heavy and lax submucous tissue, and of the true vocal cords. As one approaches the laryngeal ventricles and trachea, the previous coating is replaced by delicate ciliated epithelium. The mucous membrane in the neighborhood of the laryngeal ventricles is itself very loosely attached, exceedingly thin, and frequently thrown into folds on the true vocal cords. Acinous glands are here very abundant, being fifteen to twenty to the square cm., and arranged lengthwise. Around the ventricles they are very numerous, and their outlets are lined with cylindrical, rarely with ciliated epithelium. The true vocal cords are not supplied with glands of any kind.

The acinous glands have no lymphatics leading into them, but the latter may be seen in other parts of the mucous membrane of the larynx and submucous tissue. In

fact, they are both large and numerous, and have the general character of lymphatics, the endothelium in particular. In the epiglottis of the new-born, they form but a single layer, in the larynx and trachea two layers, and in certain parts which are covered by a considerable amount of sub-mucous tissue, there are even three layers. In the innermost, the lymphatics are arranged perpendicularly to the surface; in the outer, horizontally.

Conglomerate glands, frequently found by Verson in the cat, and invariably in the laryngeal ventricles of the dog, are seen in but few parts of the human air-passages. The exceptions, such as the vestibule of the larynx, the borders of the ary-epiglottidean folds, and the posterior surface of the epiglottis, which were noted by Luschka, rather tend to emphasize the general rule than to disprove it.

THE MUCOUS MEMBRANE OF THE TRACHEA AND BRONCHI

contains more elastic than fibrous tissue, a moderate amount of lymphatic vessels, no lymphatic glands, an abundance of mucous glands, and is freely supplied with ciliated epithelium.

The following table will serve as a résumé of the above-described anatomical characteristics.

Among all the tissues and organs thus far spoken of, the elastic, which is an important element in the formation of connective tissue, is least affected by chemical or organic influences. It does not become glutinous by boiling, and is not decomposed by water, acetic acid, alcohol, the gastric juice, or moderate heat. It is dense, firm, and hard, and possesses a degree of elasticity that no other tissue has attained in a normal condition. It has but few blood-vessels, no nerves, few lymphatics, and undergoes a very slow metamorphosis. When injured, it is never reproduced, its reparative process results in the formation of a fibrous cicatrix. Now, elastic tissue is not present in the mucous membrane of the nose, it is so to a considerable

	MOUTH.	TONSILS.	NOSE.	EPIGLOTTIS.	LARYNX.	TRACHEA AND BRONCHI.
Elastic fibres.	Many, mixed with connec. tissue.	Copious.	None.	Many.	Muc. membrane thin, sometimes folded on true cords.	Many.
Connective fibres.	Numerous.	Copious, on surface and in follicles.	Exclusively.			Less.
Epithelium.	Thick, pavement.	Pavement.	Pavement in cart. portion, cylindrical in resp., fimbriated in olfact. portion.	Pavement, at insertion cylinder (with fimbriated).	Pavement on true cords and ary-epigl. fold, fimbriated towards ventricle and trachea.	Fimbriated.
Lymph-vessels.	Frequent on lips, tongue, soft palate, pillars, cheeks, most on uvula.	Lymph corpuscles, no ducts.	Very numerous in lower, scanty in olfact. portion.	Few anteriorly, less posteriorly.	Not on true cords, none in the ac. glands near vent.	Some, no lymph glands.
Blood-vessels.	Moderate.	Few.	Very abundant.	Few.		
Acinous muciparous glands.	Frequent, most on soft palate.		Very numerous in resp. portion.	None.	Frequent round ventr., with cyl. epithelium in ducts, without lymphatics, none on true cords.	Very numerous.
Nerves.	Moderate.		Very numerous in resp. portion.			

amount in the buccal cavity, very abundant in the walls of the lymph-follicles of the tonsils, and so prominent an element in the trachea that the fibrous tissue is relatively trifling. The influence of the anatomical condition on the diphtheritic process must be very marked. It can easily be demonstrated that where the elastic tissue is present to a large amount, an antagonism to diphtheritic impregnation is maintained for a long time, but when it is obliged to yield, there is a corresponding resistance to recovery.

It is the pavement-epithelium, according to Wagner, which gives the first foot-hold to diphtheritic membrane. Where it is most abundant, the diphtheritic poison can most easily settle and develop. Thus it is that the tonsils, not from their prominent situation alone, but from the character of their surface also, are favorable to the reception and further development of the infection, and their elastic and connective fibres, when once affected, are apt to harbor the process a long time. Ciliated epithelium, on the other hand, is not so liable to be affected. It occupies a higher rank in the scale of animal formation, has a more complex function and a greater power of resistance.

The presence of a large number of mucous glands impedes, as a rule, by the presence of the normal secretion, an extensive destructive action upon the tissues. The secreted mucus assists in removing epithelial masses, and even fibrinous exudations, from the surface. The tissues themselves do not take an active or prominent part in the process; the serum of the mucus penetrates the parts which are the seat of morbid deposits, and tends to predispose them toward maceration, and the mucous secretion raises mechanically the superjacent deposits from their bed. Thus it is that the deposits in the respiratory portion of the nasal cavities are frequently cast off through the nostrils, probably because they have been produced in excess, and in a similar manner, the membranes that have formed in the trachea are ejected in a semi-solid con-

dition through a newly-made tracheotomic outlet. The large number of mucous glands in the larynx and trachea is unquestionably the reason why the lymphatic vessels of the mucous membrane are not influenced by the overlying loosened masses, and will not absorb; hence laryngeal and tracheal diphtheria have decidedly a local character, and are so frequently devoid of constitutional symptoms.

THE VOCAL CORDS

deserve especial notice. They form the borders of the narrowest aperture of the air-passages. Foreign bodies, whether malignant or otherwise, are detained or retained by them. They are covered with pavement epithelium which, as has been remarked, is the principal resting and breeding place of the diphtheritic affection. They have no muciparous follicles, and few or no lymphatic vessels, and therefore if there is any part which is predisposed to diphtheritic infection it is certainly the vocal cords. Where the poison is insufficient for general infection, it is at times capable of still producing local phenomena. Where an epidemic of diphtheria has died out, a local diphtheritic infection can still take place, and individual cases occur now and then with an almost insignificant power of infection. Such occurrences take place for years or decades, and give rise to the so-called sporadic membranous croup, in the same way as we have for years heard of an occasional case of sporadic cholera or of a few cases of small-pox. There may not be sufficient infectious material to act on the blood, larynx, or pharynx, but just enough to gain a foothold on the prominent vocal cords with their pavement epithelium.

On the other hand, the absence of acinous glands on the vocal cords must serve to a certain degree as a guard against the disease. Dry, atrophic, smooth conditions of the mucous membrane of the fauces likewise tend to ward off an attack of diphtheria. A more or less moist or

viscid condition of the surface is necessary in order that the poisonous material may cling thereto. The comparative dryness of the vocal cords, however, considered by the side of the perpetually moist and uneven surface of the pharynx, would not appear as favorable to the deposition of foreign infectious elements. Thus there are certain conditions predisposing to, others antagonizing infection. They demonstrate, however, why laryngeal croup is more frequent in winter than in summer, in direct proportion to the greater frequency of laryngeal catarrh in winter than in summer. Diphtheritic membranes on the vocal cords are not easily cast off, for there are no subjacent muciparous glands whose secretion could wash them away. No general infection can arise from them, for they have no lymphatic vessels which could serve as carriers of the poison; furthermore, suffocation occurs too early to enable the few neighboring lymphatics to absorb and transport the poison elsewhere, in case the deposits should finally become macerated.

It strikes me that these anatomical and physiological considerations will help to throw light on the question of the mooted difference between "croup" and "diphtheria." The lymphatic and vascular systems must be looked upon as the most influential factors in the development and severity of the general phenomena in diphtheria. The absence of the lymphatics and the paucity of blood-vessels explain why diphtheria of the tonsils has so mild a character. The large number and size of, as well as the direct communication of the lymphatic ducts of the Schneiderian mucous membrane with the lymphatic glands of the neck accounts for the dangerous character of diphtheria of the nose. However, the direct infection, *i. e.*, the absorption of the poison into the body, is not always dependent on the lymphatics, for they have occasionally neither enough time nor the opportunity to use their power. For instance, in those cases of diphtheria of the nose in which early and slight epistaxes occurred,

the poison appears to have been absorbed directly into the blood-vessels. Then we fail to observe the ordinary swelling of the neighboring glands of the neck, but the general symptoms are very rapidly developed. Usually, however, infection results through the lymphatics. The fluid contents of the tissues, or such particles or elements as are suspended therein, be they of a gaseous, chemical, or parasitic nature, are conducted to the lymphatic glands whose peripheric fascia propria serves as the first resting-place, for here the lymphatic vessels subdivide, previous to penetrating the fascia and evacuating their contents into the lymph-spaces of the alveoli of the cortical substance. As is well known, the latter are filled with lymph-corpuscles consisting of coarse, granular protoplasm. From hence the lymph is carried off by delicate vessels into the substance of the glands. Their structure is the same as that of the cortical substance, with the exception that they are less dense because of their containing less connective tissue and more and larger lymph spaces. All the infectious material that had entered the afferent channels and is small enough to be carried onward with the lymph and newly-suspended lymph corpuscles, is now introduced into the uninterrupted lymphatic and vascular currents, unless obstructed by a second series of lymphatic glands. All that which is as delicate as the lymph, and not larger in its microscopic proportions, is conveyed without interruption through the fascia propria into the reservoir of the cortical and medullary substances of the glands. There may be two conditions, however, which will serve to impede the current. In the first place, the foreign material may be present in too large an amount to circulate with ease; the result will be stagnation and consequent irritation, either in the fascia propria or in the glandular substance. By pressure, the capillary circulation becomes interfered with, proliferation ensues, the circulating lymph mingles with the white corpuscles from the lymph spaces, and the result is an abscess in the

intra- or peri-glandular tissue. When this is not the case, the foreign material is retained in the interior of the fascias in the connective tissue or in the dilated lymphatic vessels of the cortical substance. Thus fluids injected into the cortical substance have been found collected in the external portions of the glands, where it was impossible for them to be carried into the circulation. Hence the gland may serve as the receptacle of noxious elements which have circulated in the lymphatic current, with or without danger to the integrity of its tissues. In this manner a second attack of diphtheria may often find its explanation in the absorption of stowed away poison; syphilis also, and other poisons, may be stored in the gland, or if their presence prove irritating, give rise to speedy suppuration, and even elimination, provided the abscess be opened sufficiently and early.

The glands may swell considerably, though the foreign matter be not present in excess, but of a very irritant character—this will occur where the poison is of a heterogeneous nature—no matter whether the elements are of a chemical or a parasitic nature. The swelling may be very marked. In infection from cadaveric poison, the axillary glands may become fifty times their normal size, for it is they and not the cubital glands which form the first upward station of the greater number of lymphatic vessels of the fingers.

The glands of the neck, too, may in diphtheria become enormously swollen within a few hours. Swelling to a certain degree always results when there has been an irritation of the lymphatic system. I have already referred to the irritation produced by a simple nasal or oral catarrh, resulting in a gradual swelling of the neighboring lymphatic glands. It is a positive fact that many a case of so-called scrofula, founded on an indolent swelling of the lymphatic glands, would find a ready explanation in the presence of a chronic nasal catarrh or of superficial erosions of the buccal mucous membrane. If the absorbed

material be but little irritating, but in sufficient amount and extending over a longer period, a considerable mass may be taken into the circulation before they can give rise to a local swelling. If the materials are very minute and in moderate amount, they may traverse the lymphatics for days and even weeks, and finally give rise to general infection rather than to a local disorder. This will ensue when elimination is less rapid than absorption. In this way an infectious poison, whose elements, organic or not, may be ten or twenty times smaller than the lymph-corpuscles—if it be feasible to calculate their size or predict their immediate changes—may stagnate in the smallest blood-vessels, proliferate rapidly, and then travel onward, or give rise to changes in the red blood-cells and leukocytes of the smallest vessels, and lead to deposits of a purulent, septic, and gangrenous character, and a disintegration of the normal tissues on a large scale, before local symptoms can be diagnosticated. Indeed, we often find that the apparently mildest cases of diphtheria eventually exhibit the most unpleasant symptoms. Sudden collapse and death are usually noticed in the apparently mild cases, and thus we are rarely in a position to say at the outset without hesitation (not even when nothing is perceptible except a circumscribed local diphtheria of the tonsils) that the individual case will prove mild or severe, that it will be short-lived or followed by successive ailments.

SUMMARY.

The membrane, or the granular infiltration, are characteristic of diphtheria. Its contents are, more or less, fibrin, changed epithelium, blood, mucus, and pus. The main changes take place in the pavement epithelium, according to E. Wagner. The epithelium is as rapidly renewed as changed. The views of histologists do not, however, agree about the nature or the importance of the epithelial transformations. The doctrine that the diph-

theritic process is caused, excited, or aided by bacteria is either sustained or denied by many. The membranes produced by artificial irritation are considered by some identical with, by others to differ from, the genuine diphtheritic product. The former view is held by the foremost clinicians.

Most organs are liable to participate in the diphtheritic process, the blood (thin, black), the heart (granular, fatty, hemorrhagic, thrombotic, endocarditic), the lungs (several forms of inflammation, infarctus, œdema, emphysema), the spleen and sometimes the liver (large, hyperæmic, soft), the kidneys (congested, nephritic), the muscles (ecchymotic, degenerated, atrophic), the glands (swelled, ecchymotic, gangrenous, suppurating), the intestine and other viscera.

The several forms of diphtheria have a peculiar predilection for certain organs or part of organs. This predilection depends on the character of the surface and its epithelium. The greater or less amount of elastic tissue, the number or absence of muciparous glands and of lymph vessels, the nature of the epithelium (pavement, ciliated, or fimbriated), determine the character of the membrane in the different locations. Copious secretion of mucus induces early maceration.

The vocal cords are apt to serve as resting-places for the diphtheritic poison, but constitutional infection is prevented by the absence of lymphatics, and rapid maceration by that of muciparous glands.

Nasal diphtheria is apt to be very fatal by the immense net of lymphatics in the Schneiderian membrane, or by direct absorption into the superficial blood-vessels.

Lymphatic glands swell very considerably, but suppurate but rarely. They may serve as depots from which reabsorption and relapses may take place.

CHAPTER VII.

DIAGNOSIS.

The characteristic sign of diphtheria is the membrane, with more or less injection of the surrounding parts. In regard to this more or less injection, I will say that pharyngeal congestion, when it is uniform, may or may not point to imminent diphtheria. When it is local, confined to one side mainly, it is either traumatic or diphtheritic. White spots which are easily washed away, or can be removed with a brush, or squeezed out of the follicles of the tonsils, into which a probe can be introduced sometimes to the depth of one-half inch, soon announce their true character, either a simple catarrhal secretion or the effects of suppuration. Even though the superficial deposit contain oidium or leptothrix in considerable numbers, it can easily be removed; I have only known the totally inexperienced to mistake muguet of the mouth for diphtheria. In the larynx, muguet is, moreover, very rare indeed, and always circumscribed, mainly on the true vocal cords. The gray discoloration of superficial follicular ulcerations, as observed in the ordinary form of stomatitis follicularis, can hardly fail to be recognized. Such patches are very numerous in the fauces and on the lips and cheeks, never on the gums, except in ulcerous stomatitis which is not follicular. They are accompanied, too, by vesicles containing more or less serum, which have not yet ruptured. It must be remembered, however, that the mucous membrane, when deprived of its superficial covering, is liable during an epidemic of diphtheria to become infected, like every other wound. I have seen cases in which stomatitis and diphtheria existed side by side, the latter having

invaded the exposed surfaces resulting from the former. The examination of the entire throat is not always easy. Very young children vomit frequently and persistently before the whole surface is exposed to view, and not infrequently, repeated examination with the spatula is absolutely necessary. In general, however, the slight attempts at vomiting suffice to cause a great part of the swollen posterior portion of the tonsil to revolve into view. I have heard that the pale surface of old hyperplastic tonsils has been mistaken for diphtheria; I merely mention this fact to stigmatize so gross an error. When a discoloration happens to be the result of a deposited flake of mucous, a drink of water will remove it.

Fever is not always a prominent symptom; in fact at times it is necessary to take the rectal temperature in order to discover an elevation; as a rule, simple diphtheria of the tonsils is accompanied by very little fever. Still there are plenty of exceptions. But the differences of temperature are not more striking than in most other infectious diseases, whose either mild or severe invasion may offer an obstacle to immediate diagnosis. As the height of the fever does not absolutely determine or even indicate the character of the subsequent course of the disease, but little importance is to be attached to the temperature, unless there be a very marked elevation. A sudden rise frequently occurs with lymphadenitis.

High fever in the beginning may render the diagnosis difficult or postpone it. A girl of fifteen years, who had suffered from intermittent fever a great deal previously, was taken sick with a chill, with flushed face and throat after, and a temperature of 106° . This attack also was referred to the influence of malaria poisoning, the single attacks of which had generally lasted from twelve to sixteen hours. The next morning her temperature was still 104° . The diagnosis of malaria was dropped. There was a slight patch on one of her tonsils. That very evening her temperature was still the same, and some little albumen in

her urine. A great deal of albumen the day after, and the third day but little albumen, copious urine, and a temperature of $100\frac{1}{2}^{\circ}$ (rectum).

The absence of lymphadenitis does not nullify the diagnosis of diphtheria, for when the tonsils are affected by the disease, there is frequently little or no swelling of the neighboring glands. The swelling of the glands enables us to locate the affection in a mucous membrane richly endowed with lymphatic vessels. It is very marked when the nose is affected. A few hours' duration of nasal diphtheria suffices for the development of a severe lymphadenitis, especially at the angles of the jaw. When the latter condition is found to exist, the throat should be examined with the idea of finding a membrane extending upward; nasal diphtheria is very liable to complicate an affection of the uvula and arches of the palate. The membrane cannot well be seen by looking through the nostrils; highly serviceable for this purpose is a very short, broad rhinoscope reaching upward to the bony structure of the nose. However, nasal diphtheria may frequently be diagnosticated some days before the membrane becomes visible, by the rapid development of lymphadenitis; this may be done even where the sweetish, musty odor of certain forms of diphtheria is absent. Yet nasal diphtheria may occur without much lymphadenitis; as, for instance, when the blood-vessels are very numerous and superficial, and thereby give rise to slight hemorrhages at the very beginning of the sickness. In such cases the lymphatic vessels are little, if at all, required to transmit the poison, the open blood-vessels replacing them in the function of absorbing. Naturally there are cases in which an ocular examination is not immediately or even at any time satisfactory. In the journals we read of brilliant results of rhinoscopic and laryngoscopic examination; in practice we see but few; the patients are less inclined or in the proper condition to submit thereto, than the observer. This holds good

especially for the dyspnœa accompanying laryngeal diphtheria where the diagnosis may be doubtful, when no membrane can be detected in the fauces; even if membrane be observed there, symptoms of suffocation may still arise from a laryngeal stenosis independent of membranous deposits in the larynx. If aphonia and difficulty of both inspiration and expiration be present at the same time, there is certainly membranous occlusion. If aphonia appear late, or even toward the very last, and only inspiration be impeded while expiration is comparatively free, there is an œdematous saturation of the ary-epiglottidean folds and its copious sub-mucous tissue, and consequently of the posterior attachment of the vocal cords. Such a condition is not at all uncommon—whereas, a general œdema glottidis in connection with diphtheria is of exceedingly rare occurrence—and has forced me to tracheotomize many times; but again, a comprehension of the true condition, where it occurred in not very severe cases, has on several occasions enabled me to avoid an operation. This local œdema may sometimes be detected by palpation in the region of the swollen posterior wall of the pharynx.

One of the diagnostic symptoms of membranous laryngitis, believed in and referred to by Krönlein, does not exist, viz., the swelling of lymphatic glands, which in his opinion is pathognomonic. Not only is that not the case, but what I said above of the absence or scarcity of lymphatics and muciparous glands of the vocal cords and their neighborhood renders the absence of lymphatic swellings a necessity, provided the latter do not depend on complicating diphtheria in other localities. In uncomplicated diphtheritic laryngitis I expect no lymphadenitis. The character of the laryngeal membrane does not depend at all on the condition of the pharynx. The latter may have membranes of any description or consistency, without being able to determine the condition of the larynx. I lay stress on this fact because no less a writer than Krönlein believes that where there is but little or no membrane

in the pharynx, that in the larynx is rather loose and movable.

One of the pathognomonic symptoms of diphtheritic laryngitis, "membranous croup," is the relative *absence* of fever. Catarrhal laryngitis, pseudo-croup, is a feverish disease. A sudden attack of "croup" with high temperature—provided there is no pharyngeal or other diphtheria present—yields a good prognosis; without much fever, a very doubtful one. If I had but words strong enough to impress that fact upon the minds of my readers, for this is the very diagnostic point against which most sins are committed. A boy of four years, F. M., in the practice of Dr. Teller, had in December, 1877, an attack of tonsillar diphtheria with very little fever; after a few days, his diphtheria being better, fever set in (104° – 105°) with hoarseness and some stenosis. It proved a temporary affair of short duration (catarrhal laryngitis), of which he soon got well. On January 3d, 1878, moderate laryngeal stenosis, hoarseness; supra-clavicular and diaphragmatic exertion not great; inspiration a little prolonged, with almost a normal pulse; a slight diphtheritic spot on lower lip, and the end of the tongue, and a temperature (rectal) of 101° . Diagnosis: membranous laryngitis, which was verified by the experience of the following days and necessitated tracheotomy.

Another boy, three and a half years old, H., a patient of Dr. Obbarius, exhibited at 6.30 P.M., on November 2d, 1877, the following symptoms: Slight redness of fauces, hoarseness, difficulty of both inspiration and expiration, pulse rather normal. *No elevation* of temperature. Diagnosis: laryngeal diphtheria ("membranous croup"). At 2 A.M., November 3d, tracheotomy had to be performed hurriedly, while the temperature was $99\frac{1}{2}$ (rectal), pulse 96 immediately after the operation, which produced quantities of false membrane.

Membranous deposits inside the larynx are characterized by the above symptoms; also the paralysis of the vocal

cords produced by ary-epiglottic œdema, posteriorly, by the symptoms enumerated above. Now and then there is a complication of both, now and then the symptoms are not well pronounced. Sometimes, when there are membranes on the tonsils, it may be of importance to watch the posterior aspect of the pharynx. When it is not swelled, not œdematous, the stenosis is probably of a membranous character. When it is œdematous, the probability is in favor of œdema about the insertion of the vocal cords.

L. Fleischmann has placed the principal real or alleged symptoms of "croup" and diphtheria side by side in the following manner :

<i>Croup.</i>	<i>Diphtheria.</i>
Not contagious.	Contagious.
True pseudo-membrane lying on the surface of the mucous membrane, from which it can be removed.	Never a true croup-membrane, but deposits consisting of degenerated and exfoliated epithelium, fungi, and detritus.
Most in children.	Occurs alike at all ages.
Most frequently affects the mucous membrane of the air-passages.	"Multilocular invasion" frequently, the fauces, nose, genitals, intestines, and the skin being affected simultaneously.
Paralysis never occurs.	Even in mild cases severe nervous disturbances.
No infection of the blood, with corresponding symptoms depending thereon.	Infection of the blood and fatty degeneration of the striped muscular tissue, especially that of the heart.
Swelling of the glands, but almost never suppuration of fœtid character.	Suppuration of the glands of frequent occurrence.
Begins as a catarrh that follows immediately after infection.	Has a period of incubation and prodromi.

Croup.

Croup may run its course without diphtheria.

Not inoculable.

J. Solis Cohen likewise tabulates the clinical differences between the two in a concise form, after acknowledging that there is no actual anatomical distinction between croup and diphtheria, either in the morbid products or the subjacent mucous membrane. His parallel is as follows:

Croup.

Not specific in its origin.

Never contagious.

Not inoculable.

Not adynamic.

Usually sporadic.

Rarely attacks adults.

Always accompanied by an exudation.

Only fatal by physical obstruction to respiration.

No weakening of the heart's action.

Pulse frequently strong and hard.

Respiration accelerated in proportion to the pulse, rarely less than 1 : 4.

Rarely albumen in the urine.

No secondary paralysis.

Tolerates antiphlogistics.

Rarely occurs more than once in the same person.

Diphtheria.

Diphtheria may run its course without croup, and invade other parts beside mucous membranes.

Inoculable.

Diphtheria.

Specific.

Frequently contagious.

Inoculable.

Adynamic.

Generally endemic or epidemic.

Frequently attacks adults.

Occasionally no exudation occurs.

Often fatal without the least impediment to respiration.

Marked weakening of heart's action.

Pulse never strong and hard, even though rapid and full.

Respiration not accelerated, usually less than 1 : 4.

Albumen frequently present in the urine.

Secondary paralysis frequent.

Does not tolerate antiphlogistics.

Frequent relapses.

I gladly devote some space to these attempts at simplification and explanation, in order to demonstrate to the reader the errors or exaggerations contained therein. To discuss the individual points separately would certainly be superfluous, after the consideration of the subject in which the reader has thus far accompanied me. These tabulated comparisons are not even convenient. Aside from the positive errors which they contain, hardly a single case of either "croup" or "diphtheria" could be appropriately placed beneath either head. Conditions which are dependent upon each other and which even clinically blend into each other continually, of which one indeed (croup) is recognized at last as a purely clinical term (unless the term croup be only applied to pseudo-membranous stenosis), cannot be arbitrarily tabulated.

To the above differential symptoms, Lyon (Trans. Conn. St. Med. Soc.) adds the following :

<i>Croup.</i>	<i>Diphtheria.</i>
Pseudo-membranes of the skin never observed.	Pseudo-membranes of the skin occasionally observed.
Generally in cold weather.	Is little influenced by weather or season.
The larynx the principal seat of the disease.	The principal seat of the disease above the larynx.

The first distinction does not exist, as any one knows who observed croup beyond its suffocative symptoms. Tracheotomy wounds, though carefully joined, frequently become diphtheritic within twenty-four hours, and from thence I have seen an extension of the process to the skin, and anywhere. Its author himself dulls the edge of the second distinction by his "generally" and "little," and in his anxiety for localization, he has in the third distinction simply reiterated that which has been assumed as a matter of convenience, namely, the designation, by the term "croup," of the pseudo-membranous deposits in the larynx, which give rise to stenosis. But,

this is not a polemic book, and I therefore refer my readers to former pages.

PRIMARY DIPHTHERIA OF THE TRACHEA AND ASCENDING
CROUP

does not occur frequently, yet it can be diagnosticated, and actual observation on the living and dead contradicts flatly the opinion of many writers whose opportunities may have been limited. Quite lately (Klebs, *Handb. d. Pathol. Anat.*, VII., p. 283, 1880), Hans Eppinger risks the statement that "croup and diphtheria occur also in the trachea, but only when descending from the larynx." Without (occasionally with) an affection of the fauces, without general symptoms that would cause a feeling of anxiety to the parents, without more fever than one would expect in the slightest bronchial catarrh, without much dyspnoea, and after symptoms of a slight bronchial or tracheal catarrh which have lasted a few hours or days, the little sufferers are most abruptly attacked by a stenosis of the larynx. Within an hour, or even less time, they become cyanotic; tracheotomy affords but a slight temporary relief or none at all; and the entire process occupies a very short space of time. Rarely is a large amount of membrane found in the larynx, but very much in the trachea and its larger ramifications. The disease began there, and without causing occlusion, because of the large size of the organ, ascended to the larynx, where it gave rise to a far more speedy death than is usually brought about by a descending croup. I have operated perhaps fifteen times in such cases, and no case, from the commencement of urgent symptoms to death, ever lasted more than a day, but many a few hours only.

The secondary descending diphtheria of the trachea can only be recognized after tracheotomy has been performed. If an elastic catheter, feather, or probe be passed through the tube, the slightest contact with mucous membrane that is not covered by deposits will give rise to coughing.

In proportion to the depth to which the process has descended, with a corresponding deposit of membrane, will this effect become less noticeable. By degrees the irritation may be applied one or two inches and more below the inferior extremity of the tube, without producing any reflex phenomena. The approach of new symptoms of suffocation (which do not assume the fearfully violent character of laryngeal stenosis) and cyanosis, gradually usher in death which can in no wise be warded off. Although the duration of this scene must vary in different cases, yet I can point to a series of cases in which the interval between the performance of tracheotomy (which I undertook at the time deemed most proper by myself, and hence not late) and final death was sixty hours.

PNEUMONIA

accompanying the general process can be diagnosticated only if the larynx be not much affected. The latter, however, is usually the case, and the laryngeal râles in such a case drown the auscultatory signs of pulmonary inflammation; percussion, too, gives no satisfactory results, as the dulness may be caused as well by collapse of the lung-tissue as by infiltration. The same may be said of bronchitis and acute œdema which may be looked upon as the direct results of rarefaction of the air in the bronchi and alveoli of the lungs. Rapid increase of temperature, together with increased number of respirations, speak for pneumonia.

Diphtheria of the VAGINA, of the CONJUNCTIVA, and of WOUNDS can only then be confounded with a simple purulent coating when an ocular examination is the only means at our command; even then rarely. The same holds true for intestinal diphtheria. Large shreds and cylindrical moulds are not always formed of diphtheritic membrane, but sometimes of mere mucus compressed into that shape, with little epithelium and almost never with blood. Thus they are seen in chronic catarrh of the colon. In the

dysenteric form of intestinal diphtheria the thrown off shreds are generally not large, and easily recognizable together with their accompanying symptoms.

THE ERUPTION

occurring in the course of diphtheria appears first on the warmer parts of the body, as the chest, neck, and abdomen; occasionally, however, it covers the entire body, and is distinguished from the scarlatinous eruption in that the latter more frequently appears first on the hips and extremities. Where it covers the entire body at once, in scarlatina, there are more severe general symptoms and higher fever than in diphtheria. In the former, the eruption lasts from five to six days; in the latter, but a few days. Still mistakes may occur, as the intensity, extent, and duration of the eruption in scarlatina may be very variable. In general, however—and this fact is of value in the diagnosis, to a certain degree—a marked scarlatinous eruption in the earliest period of the disease is accompanied by a more characteristic erythema of the mouth and throat than in diphtheria, and with less diphtheritic deposits. These will, in scarlatina, appear after a few days as a rule, and not in the beginning. In diphtheria, the characteristic symptoms belonging to the tongue, redness, throwing off of the epithelium, papillar elevations, etc., are not so well marked. At times, however, the character of the desquamation only will decide the nature of the efflorescence. It appears, however, that in some epidemics diphtheritic eruptions are but seldom observed, while in others they are more frequent.

The appearance of

ALBUMEN

in the urine will serve as a valuable diagnostic point, sometimes, between diphtheria and scarlatina. In the latter disease, it is rarely noticed in the first week; it generally appears about the ninth or tenth day at the earliest, and it may be delayed until the twenty-fifth, even to the thirty-

third in my experience. When it appears in the first week, it not infrequently presents the picture of a dangerous form of general diffuse nephritis, which is apt to terminate lethally. In diphtheria, albumen is pretty sure to appear, if at all, within the first few days, neither the degree of fever nor other general symptoms affording an explanation of its presence. It is frequently found, and in large quantities too, for a day or two, apparently as a symptom of rapid elimination, in cases which set in with a high fever, which lasts but a short time, and gives way to almost complete apyrexia.

GANGRENE manifests itself in a destruction of the tissues, for instance of the vagina or cornea, and depends sometimes on pressure by the impregnated surface; or it occurs on such privileged localities as are adapted, from their coating of pavement epithelium, for deep inroads of the degenerative process. Still the genuine necrosis of the tissues occurs in other parts of the mouth besides the tonsils, but we must be careful not to declare any thick black briny masses at once to be gangrenous. Not infrequently they are merely deposits which are easily removed. Genuine gangrenous masses readily bleed, either from the sharp corroded edges or from deep-seated vessels which have been injured. I have but rarely seen dangerous hemorrhages from gangrenous portions of the neck, and not many deaths therefrom.

EPISTAXIS

is not infrequently a formidable symptom. Its seat can sometimes be estimated by the facility with which, in different postures, the blood makes its appearance in the throat.

DIPHTHERITIC PARALYSIS

exhibits certain peculiarities which facilitate diagnosis. The latter presents no difficulties for the practitioner who has witnessed the entire course of the disease and sees

paralysis appear during convalescence. It becomes all the more easy when the soft palate is the first to be attacked, and is gradually followed by the implication of other parts. But in those cases where the diphtheritic process was not observed, and the soft palate was not affected or became so only later, the diagnosis will be more difficult, and may even be involved in utter darkness. The knowledge of the fact that diphtheria has pre-existed may arouse suspicion and guide the physician to a proper appreciation of the case. But it is necessary carefully to weigh the accompanying circumstances. A migrating paralysis of mixed character, like the diphtheritic, may be either peripheric or central. It is important to determine this point in the first place. Severe central lesions, whether of diphtheritic or other origin, will invariably present a certain clinical picture. Difficulties arise only when the question of multiple lesions of a different character, such as hemorrhages, sclerosis, can be raised at all. The more frequent, if not the constant, form of diphtheritic paralysis is the peripheric; it runs a precisely opposite course to that which is described by some authors. It is not the assumed regularity, but just the remarkable irregularity and wavering character of the paralysis, together with a capriciousness of the symptoms and of the affected organs or parts thereof, which are especially characteristic of the disease.

As a general thing, the paralytic symptoms commence in the soft palate, and pass to the ciliary nerves after (mostly bilaterally). But the reverse may take place. The symptoms, when the patient does not get well soon, may extend over months, or rather, one will be replaced by another. Beside the above mentioned, there may be strabismus, general debility of the muscular system, local paralysis, atrophy of single muscles, atrophy of skin, and nutritive disorder with alopecia, disorder of sensibility, not always of a subjective character only, dyspnoea from either degeneration of heart or paralysis of respiratory

muscles, irregular behavior under the influence of faradic (diminished) and galvanic (normal, sometimes increased, diminished after a while) currents. All the time, however, the sphincters are intact (with very rare exceptions) as in the amyotrophic or peripheric paralysis of children.

SUMMARY.

Diphtheria is characterized by its membrane. The diagnosis from muguet is easy. Complications with follicular stomatitis are of occasional occurrence. Follicular inflammation of the tonsils is recognized by its local character, by the ready removal of the deposits, and the easy introduction of a probe into the follicle. The congestion in the diphtheritic pharynx is sometimes less pronounced than in catarrhal pharyngitis. In the latter the hyperæmia is general, in the former it may be local.

Fever is not always high. Sometimes the temperature is even low in very bad septic cases. High temperatures in the beginning are less frequent than, for instance, in scarlatina. Glandular swelling may be absent for many reasons.

Nasal diphtheria has much glandular swelling; may, in some distinct cases, have none at all.

Diphtheritic laryngitis has less fever than catarrhal laryngitis, and when uncomplicated shows no glandular swelling. The character of the laryngeal membranes does not depend on the condition of the pharynx. Complete aphonia and uniform difficulty of inspiration and expiration indicates membranous obstruction; difficult inspiration with easier expiration and but partial hoarseness or almost clear voice indicates the presence of local œdema and consecutive paralysis of the vocal cords.

Primary diphtheria of the trachea is difficult to diagnose; it is likely to exist when after apparently catarrhal symptoms those of laryngeal stenosis occur very suddenly and fatally. The progress of the diphtheritic process downwards can be watched through the trache-

otomy tube and estimated by the absence of irritability of the mucous membrane of the trachea.

The diagnosis of pneumonia accompanying laryngeal diphtheria is not impossible. In the other forms of diphtheria it is recognized by its usual symptoms.

The cutaneous eruption of diphtheria is usually distinct from scarlatinous eruptions, and the diagnosis easy in most cases. Albuminuria is mostly an early symptom, and disappears more readily than in scarlatina.

Diphtheritic paralysis is recognized by the previous history of the disease, by the frequency of its starting from the pharynx, its irregular course, its mostly peripheric character, and the absence of symptoms belonging to bladder or rectum. It is mostly motory, sometimes sensory or sensitive.

CHAPTER VIII.

PROGNOSIS.

Trousseau once expressed his opinion that diphtheria was more dangerous than cholera, yellow fever, or the plague. He certainly said so under the influence of the impression conveyed by some of the worst septic cases we are apt to meet with. Fortunately the sad picture is greatly overdrawn. I have already, on some previous pages, alluded to the fact that the majority of cases are of a mild type, and that in many a season the ratio of mortality is but small. Many a year it was not higher than five per cent of all the cases. Ten per cent is certainly a high rate. Still, as far as each individual case is concerned, there is hardly a disease in which the prognosis is more uncertain than in diphtheria. Before the process has fully run its course, it is unjustifiable to consider the favorable termination secured; even when it is completed, a relapse may occur, which again casts obscurity over the entire question. The general character of individual epidemics, now mild, now severe, permits, it is true, to rely to a certain extent on probabilities, but the physician will often enough be deceived, and more frequently, too, in mild than in bad cases. There is a certain class of cases in which the prognosis is absolutely unfavorable; there is another class in which it appears favorable, and yet dangerous symptoms and a fatal termination ensue. In general, the prognosis is favorable when the affected surface is of small extent, and where such parts are the seat of disease as have little communication with the lymphatic system. To the latter class belongs simple diphtheria of the tonsils. Marked glandular swelling,

particularly if arising suddenly, is always an uncomfortable sign, and calls for the utmost caution, especially if the region of the angles of the jaw be speedily and markedly infiltrated. This, as we have seen, is particularly apt to occur with nasal diphtheria, whether developed primarily, accompanied by a thin fœtid discharge, or, as is more commonly the case, secondarily from an affection of the pharynx and palate in the continuity of tissue. With the appropriate local disinfection, it is neither so absolutely dangerous as Oertel depicts it, nor so assuredly fatal as Roger but a few years ago taught in his clinique, or Kohts appears to believe (Gerhardt, *Handb. d. Kinderkr.*, III., 2, p. 20, 1878), yet it is ever doubtful. With energetic treatment, most cases will get well. Diphtheria of wounds, complicating diphtheria of the pharynx, is always an unpleasant sign; that of the mouth and angles of the mouth, associating itself with a previously existing diphtheria, having an indolent course, and producing rather a deep impregnation of the tissues than a thick deposit, presents very disagreeable symptoms. Diphtheria of the larynx, whether it be of primary origin or the result of extension from the fauces, is nearly always fatal. In severe epidemics the mortality is 95 per cent. Tracheotomy, too, saves but few of those who take the disease at such a time. In fifty consecutive tracheotomies, from 1872 to 1874, I did not see one recovery. In the last few years, I have seen few good results. In average epidemics, tracheotomy will save 20 per cent. A pulse of 140 to 160, and high fever immediately after the operation, render the prognosis bad; so does absence of complete relief after the operation. An almost normal temperature the day after the operation is an agreeable symptom, but does not exclude a downward extension of the diphtheritic process, and hence cannot be looked upon as assuring a favorable prognosis. A marked elevation of temperature occurs with a renewed attack of diphtheria, or a rapidly-appearing pneumonia, and is an unfavorable symptom. A dry character of the respiratory mur-

mur, some time after tracheotomy, indicates the approach of death from descent of the membrane, within from twelve to twenty-four hours;* cyanosis likewise, whatever be its degree of intensity. Diphtheria of the trachea, which ascends to the larynx, is positively fatal.* It has a rapid course, and tracheotomy only postpones the end for a little while if at all. The general health and strength of the little sufferer have no influence whatever.

Thick, solid deposits need not of themselves render the prognosis so unfavorable as do septic and gangrenous forms. Even in the nose they are not of as serious import as the thin, putrid discharge. I have seen recovery ensue in cases where I was obliged to bore through the occluded nasal cavities with probes and spoons. Fœtid, putrid discharges are unfavorable, but in no wise fatal; conscientious disinfection accomplishes a great deal. Slight epistaxes indicate the possibility of rapid and undoubted absorption through the blood-vessels; but here, too, the final result depends on whether the disinfection be equally rapid and thorough. The same holds true for the sweetish, fœtid odor of the breath, whether of the nose or mouth, which, on one hand, demonstrates the significance of the disease, while, on the other hand, it indicates the possibility of infection by the breath.

The height of the fever is not in proportion to the danger in the individual cases; some have a favorable, some an unfavorable termination, without fever of any account. Simple catarrh of the pharynx and larynx frequently begin with a sudden and marked rise of temperature;

* R. W. Parker (Tracheotomy in Laryngeal Diphtheria, London, 1880) says: "The presence of membrane in the trachea in a fatal case of membranous laryngitis, after tracheotomy, must be regarded as evidence of the want of due care on the part of the surgeon in charge, just as much as would the presence of a piece of gut in the inguinal canal after herniotomy, or a calculus in the bladder after the operation of lithotomy."

I do not hesitate to express my opinion that the gentleman will modify this "somewhat absolute dictum," as he calls it himself, after some more experience.

diphtheria in the same parts but rarely. There are cases, however, in which the height of the fever and the deposited membranes are in inverse proportion to each other. In these cases, the fever may subside rapidly, owing to a speedy elimination of the poison. Young children only are in danger of death from convulsions, or a rapid tissue degeneration. If the temperature rise suddenly after days of sickness, either a complication or a fatal termination is to be apprehended. Yet, if we except laryngeal and tracheal diphtheria, there are as many deaths with comparatively low, as with very high temperatures. Whether collapse has resulted rapidly or slowly, the patient dies often with low temperature. Thus a rapid elevation is hardly a more unfavorable sign than a rapid fall. The pulse, too, may be very variable. True, a small, rapid, and irregular pulse is always unpleasant, because it indicates a weakening of the cardiac function; yet, as long as it retains an approximately normal relation to the frequency of respiration, a rapid pulse gives no cause for alarm. Moreover, the pulse is not always rapid when the strength gives way. It occasionally becomes slower, and sometimes very slow, and may then become a dangerous symptom. It seems to me highly probable that there is in such cases, as in certain forms of chronic fatty metamorphosis of the heart, a degeneration of the cardiac ganglia of the sympathetic.

Every complication adds to the danger. Bronchitis and pneumonia are not infrequent; yet I have seen cases of laryngeal diphtheria recover, in which I had suspected pneumonia before performing tracheotomy, and was enabled to diagnosticate it after operating. Albuminuria in the early part of a diphtheritic attack (with high fever) is of little significance; nephritis, later in the course of the disease, partakes of the character of scarlatinous nephritis; cases of acute diffuse disease are fortunately infrequent, and the remainder are very submissive to treatment. The cases complicated with endocarditis, in my practice,

ended fatally. An early implication of the sensorium, not dependent on pressure upon the jugulars by greatly swollen glands, is an unfavorable symptom. Purpura, with profuse hemorrhages and a livid hue of the skin, is ominous; icteric discoloration, together with marked glandular and periglandular tumefaction, is absolutely fatal. Calimani observed an epidemic in which he lost one hundred and fifteen cases out of two hundred. Before the diphtheritic eruption on the integuments took place, he often noticed a cyanotic discoloration and an offensive secretion of the last phalanx of the big toe, sometimes of other toes, or of the middle finger. All of those showing these symptoms died. A case of acute diphtheritic infection with fatal termination, taking its course with the symptoms of pernicious icterus, was but lately recorded by Becker (*Berl. klin. Woch.*, Nos. 30 and 31, 1880).

CHAPTER IX.

TREATMENT.

GENERAL REMARKS.

Every case should be treated on general principles; thus it is not possible to lay down a routine treatment for every individual case. High fever should be reduced by sponging and baths, quinine, and sodium salicylate; collapse speedily treated, and severe reflex symptoms, as vomiting, etc., checked at once. Whether to employ for this purpose ether, wine, cognac, champagne, or coffee, must be decided by the physician in individual cases. The administration of the remedy, whether by mouth, by injection into the bowels, or subcutaneously, as I have employed cognac, ether, alcohol, and camphor dissolved in ether or alcohol, in some cases with decided and rapid success, must depend on the condition of the organs and on the urgency of the case. At all events, it may be stated that all the above remedies are frequently of no service because they have been administered too late, and in too small doses, and hence we may infer that to obtain the proper results both from external and internal treatment, the remedy must be employed early and often, and in sufficient quantity. If I have ever had cause to feel contented with the results of treatment in diphtheria, it is owing to the fact that I did not lose time. Moreover, the nourishment of the patient is a matter of very great importance, and should not be neglected, and no medicines resorted to which are apt to derange the digestion of the patient. It is true that caution must be exercised in the food administered to febrile patients, but we must bear in

mind that, when the lymphatic vessels are kept empty, and no new and proper material is introduced into them, the absorption of locally existing poisonous substances is proportionately increased.


I dwell particularly on the foregoing remarks for the reason that, in diphtheria, unlike certain diseases having a typical course and those of a simple inflammatory character, expectant treatment should not be indulged in. Oertel's advice, that when neither high fever nor complications are present we should quietly wait, and "act only when new and most alarming symptoms present themselves," is decidedly perilous. A mild invasion does not assure a mild course. Never has a "perhaps superfluous" tonic or stimulant done harm in diphtheria, but many a case had a sad termination because of a sudden change in the character of the disease, putting the bright hopes of the physician to shame. Only the philosopher may be a passive spectator, the physician must be a guardian. When I again read, in the work of the same meritorious author, "that when in exceptional cases, in children and young people, death is imminent, not from suffocative symptoms in the larynx and trachea, but from septic disease and blood-poisoning, it is necessary to resort to powerful stimulants," it strikes me that he is frequently too dilatory with his remedies, and furthermore, that his experience concerning the terrible septic form of diphtheria, which is so frequently met with in some epidemics, must have been very limited at that time. In New York, during the past twenty years, for every death from diphtheritic laryngeal stenosis, there have been three from diphtheritic sepsis or exhaustion. To generalize from a few cases or years would be unsatisfactory. But few authors have displayed the unselfishness of Krieger, who, in his "Etiological Studies" (Strassbourg, 1877), had repeated opportunity for observation, yet in his careful essay on the "Predisposition to Catarrh, Croup, and Diphtheritis," refers to the insufficiency of his own observations.

When a modern writer (Ripley, *Med. Rec.*, July 31st, 1880) teaches that "diphtheria is a self-limited disease," "which runs its course from a few hours to weeks," and may "end in recovery, according to the character of the epidemic and idiosyncrasy of the patient, even without medicine," he certainly stretches the definition of a self-limited disease to undue proportion, while in regard to spontaneous recovery he states what may be said of any and every disease. His teaching that the only rational plan of treatment of diphtheria is a symptomatic one, is dangerous, because it is apt to seduce into the neglect of preventives, and of the timely resort to medication, to say the least. It is true that the results of no treatment cannot be uniformly successful, but at all events the indications for causal treatment are commencing to be known at last. In that respect we have progressed somewhat beyond the most thoughtful therapeutics of the disease, as developed in the course of the last century, and so well illustrated by the "*Tentamen medicum inaugurale de cynanche maligna*," by Thomas Wilson, *Edinb.*, 1790. He says, p. 24: "*Cum hactenus nullum inventum est remedium quod contagionem in corpus receptam suffocare possit; cum medicamenta pleraque quæ putredinem corrigere dicuntur, corpus ejusque functiones manifesto roborant; et denique, cum hunc morbum comitantur virium prostratio, et, etiam ab initio, summa functionum debilitas, qualis evacuantia omnigena prohibet, indicationem curandi unicam, scil. debilitatis effectibus obviam ire, proponam. Hinc corporis conditioni obviam itur præcipue tonica et stimulantia administrando.*" (As no remedy has yet been found which can extinguish the contagion after it has been received into the body, as most medicines which have the reputation of correcting putrefaction, are roborants for the body and its functions, and lastly, as this disease is attended with great prostration and such debility of functions as to preclude the use of all sorts of evacuants, I propose but this one indication for treatment, viz.,

to meet the effects of debility. This is fulfilled by the administration of mainly tonics and stimulants.)

While speaking of stimulants, I will say a few words in regard to the dose to be given. There is more danger in diphtheria from giving too little than too much. When the pulse begins to be small and frequent, they must be administered at once. A three-year-old child can comfortably take thirty to one hundred and fifty grammes of cognac, or one to five grammes of carbonate of ammonia, or a gramme of musk or camphor in twenty-four hours. In the septic form especially, the intoxicating action of alcohol is out of the question, the pulse becomes stronger and slower, and the patient enjoys rest. In those cases in which the pulse is slow, together with a weak heart's action, the dose can hardly be too large. The fear of a bold administration of stimulants will vanish, as does that of the use of large doses of opium in peritonitis, of quinine in pneumonia, or of iodide of potassium in meningitis or syphilis. I know that cases of young children with general sepsis commenced immediately to improve when their one hundred grammes of brandy was increased to four hundred in a day.

The remarks I have made in reference to the general treatment of diphtheria naturally render superfluous a discussion of the value of abstraction of blood. To be sure, it could only be a question of local bleeding. For nobody would dare to resort to jugular venesection, as our predecessors did in the last century. It may be safely asserted of the latter that it has no influence on the process, but frequently increases the local swelling and makes the patient more anæmic. There is no case in which a resort to it would not be criminal. I can distinctly recall the time when bleeding and calomel formed the groundwork of the treatment. Until the year 1862, the death rate in Rupert, Vermont, from diphtheria was ninety per cent, according to the reports of the local physicians, and particularly of my pupil, Dr. Guild, who at that time



finished his studies in New York and commenced practising. When, in the same epidemic, bleeding and calomel were replaced by stimulants and iron, with the chlorate of potassium, ninety per cent recovered.

That attention must be paid to the general condition, mainly during a retarded convalescence from previous sickness, is self-evident. Any complications, too, must be subjected to early treatment. Diarrhea must be mentioned among these; it reduces the patient's strength very quickly; likewise, the early appearing nephritis which may suddenly end life.

One important axiom must be borne in mind, namely, that prevention is more easy than cure. I do not refer simply to the removal of the healthy members of the family beyond the danger of infection, or to the isolation of the patient. If the latter becomes necessary, the first indication is his removal to the top floor of the house. There are, in addition, however, certain prophylactic measures which will prove valuable in the hands of every good physician. It is necessary under all circumstances that the mouth and pharynx of every child be constantly kept in a healthy condition. Eruptions of the scalp must be treated at once, and glandular swellings of the neck caused to disappear. But lately some cases of laryngeal diphtheria have been traced directly to the presence of suppurating bronchial glands, with or without perforation (Weigert, in *Virch. Arch.*, Vol. 77, p. 294, 1879). The same rule applies to nasal and pharyngeal catarrhs, the treatment of which should be begun in warm seasons, when general or local remedies yield better results. Enlarged tonsils should be resected, or, where that cannot be done, scraped out with Simon's spoon, at a time when no diphtheritic epidemic is raging. It is important that this take place at a time when, even though sporadic cases of diphtheria occur, the danger of infection is not great; for, during the height of an epidemic, every wound will give rise to general or local in-

fection. This holds true for wounds of any part of the body, as well as of the mouth. I therefore avoid an operation at such a time, provided it can be postponed.

In this connection I shall speak of a remedy which I class among the prophylactic agents, namely the *chlorate of potassium*, or the *chlorate of sodium*. I cannot say that I rely on either of these remedies as curative agents in diphtheria, and yet I employ them in almost every case. The reason lies in the fact that the chlorate is useful in most cases of stomatitis, and thereby acts as a preventive.

There are very few cases of diphtheria which do not exhibit larger surfaces of either pharyngitis or stomatitis than of diphtheritic membrane. There are also a number of cases of stomatitis and pharyngitis, during every epidemic of diphtheria, which must be referred to the epidemic, sometimes as kindred diseases, and sometimes as introductory stages only, which, however, do not, or do not yet, show the characteristic symptoms of the disease.

When, in 1860 (Amer. Med. Times, Aug. 11th and 18th), I wrote my first paper on diphtheria, I based it upon two hundred genuine cases, and at the same time enumerated one hundred and eighty-five cases of pharyngitis, which I considered to be brought on by epidemic influences, but which, the membrane being absent, could not be classified as *bona fide* cases of diphtheria.

Such cases of pharyngitis and stomatitis, no matter whether influenced by an epidemic or not, furnish an indication for the use of chlorate of potassium. They will get well with this treatment alone. The cases of genuine diphtheria, complicated with a great deal of stomatitis and pharyngitis, also indicate the use of chlorate of potassium; not as a remedy for the diphtheria, but as a remedy for the accompanying catarrhal condition in the neighborhood of the diphtheritic exudation. For, it is a fact that, as long as the parts in the neighborhood of the diphtheritic exudation are in a healthy condition, there is but little danger of the disease spreading over the sur-

face. Whenever the neighboring surface is affected with catarrh, or inflammation, or injured, so that the epithelium is loose or thrown off, the diphtheritic exudation will spread within a very short time. Thus chlorate of potassium or sodium, the latter of which is more soluble and more easily digested than the former, will act as a preventive rather than as a curative remedy. Therefore it is that common cases of pharyngeal diphtheria will recover under this treatment alone, nothing else being required.

The cases of diphtheria in which the exudation is limited to the tonsils are by no means dangerous, for the lymphatic communication between the tonsils and the rest of the body is none at all, or very trifling. Thus no absorption into the circulation can take place from a tonsillar diphtheritis alone. The surrounding stomatitis and pharyngitis will be favorably influenced by the administration of chlorate of potassium or sodium, and thus the entire disease will run a favorable course, inasmuch as the tonsillar exudation will be removed within three or six days. The surrounding portions of the mouth and fauces, meanwhile, having been put into a tolerably healthy condition, the danger is passed. These are the cases which have given its reputation to chlorate of potassium as a remedy for diphtheria.

The dose of chlorate of potassium for a child two or three years old should not be larger than half a drachm (2 grammes) in twenty-four hours. A baby of one year or less should not take more than one scruple ($1\frac{1}{4}$ grammes) a day. The dose for an adult should not be more than a drachm and one-half, or at most two drachms (6 or 8 grammes), in the course of twenty-four hours.

The effect of the chlorate of potassium is partly a general and partly a local one.

The general effect may be obtained by the use of occasional larger doses; but it is better not to strain the eliminating powers of the system. The local effect, however, cannot be obtained with occasional doses, but only by

doses so frequently repeated that the remedy is in almost constant contact with the diseased surface. Thus the doses, to produce the local effect, should be very small, but frequently administered. It is better that the daily quantity of twenty grains should be given in fifty or sixty doses than in eight or ten; that is, the solution should be weak, and a drachm or half a drachm of such solution can be given every hour or every half-hour, or every fifteen or twenty minutes, care being taken that no water is given soon after the remedy has been administered, for obvious reasons.

I have referred to these facts with so much emphasis, because of late an attempt has been made to introduce chlorate of potassium as the main remedy in bad cases of diphtheria, and, what is worse, in large doses.

It is Seeligmüller especially who has recommended chlorate of potassium for that purpose in a saturated solution. Sachse also looks upon a saturated solution of chlorate of potassium as a panacea, inasmuch as he did "not lose a case out of one hundred," except those, as he says, "which were hopeless at the beginning." A young colleague in our State also recommends chlorate of potassium (six drachms daily) as his sheet anchor in diphtheria (Louis Weigert, M.D., Hospit. Gaz., Jan. 16th, 1879).

Seeligmüller administers a solution of one in twenty. Of this, he gives children of three years and over a tablespoonful every hour at first—doses which amount to half an ounce in twenty-four hours; afterward every two or three hours. To children a little younger, he gives half a tablespoonful, and continues the treatment day and night. He insists upon the necessity of not adding any syrup to the solution, and also of not allowing the patient to drink within a short time after the administration of the remedy. In his opinion, the internal treatment suffices; still he advises that the solution should be used as a wash, a gargle, and also should be snuffed.

He says that the bad odor and fever, under that treat-

ment, disappear within a very short time. The number of cases which he first reported as treated successfully in this manner was fifteen. At the same time, he gave milk, broth, egg, and a small quantity of Tokay wine. These cases were published a number of years ago. Since that time he has modified his opinion to a certain extent. He says that chlorate of potassium may prove injurious, because of the possibility of the potassium acting *upon the heart*; and that, when it does, the heart's action becomes either more or less frequent, and may be intermittent. On the other hand, he directs attention to the fact that diphtheria itself will act upon the heart in a similar way; and, as soon as such symptoms occur, quinine, coffee, and wine are recommended.

Digestion may also be interfered with by chlorate of potassium, inasmuch as when acute gastric catarrh is present the remedy is not well tolerated. In such cases, smaller quantities must be given, or the drug must be discontinued altogether. In consequence of meeting with these drawbacks, he insists upon the above method of administering the remedy only during the first twenty-four or thirty-six hours. This modification he began particularly after a few of his patients *died with a sensation of burning and soreness*.

I have reported his practice so extensively, because I mean to raise my voice against it for the reason of its dangerousness.

As early as 1860, I advised strongly against the use of large doses of chlorate of potassium, but the translation of the paper I then published in the American Medical Times, which was printed in the Journal für Kinderkrankheiten, in 1861, was so defective that I am not astonished at my warning having been overlooked on the European side of the Atlantic. The treatment is dangerous because of the largeness of the doses of the chlorate of potassium administered.

Seeligmüller himself reports a case of a boy six years

of age, who died within a very short time under the chlorate-of-potassium treatment, the main symptoms being copious greenish discharges, obstinate vomiting, and collapse. The kidneys were not examined after death, but the symptoms and the resemblance of these cases to a number of others of equal nature and result, prove them to be cases of nephritis depending upon over-doses of chlorate of potassium.

Lacombe had under observation a man who took one ounce of potassium chlorate, intending to take an ounce of the sulphate of magnesium. The man died in convulsions, after having purged very freely, and the cause of death was regarded as *excessive diarrhœa*. The probability is, that it was a case of nephritis.

Isambert, in his first reports upon the effects of chlorate of potassium, published more than twenty years ago, found among its effects increased diuresis, a sensation of heaviness and dragging in the lumbar region, such as is found after the administration of large doses of nitrate of potassium.

Ferris reports a case of death from cyanosis, with absence of pulse, within a period of thirty-six hours after a tablespoonful of the chlorate of potassium was taken.

He found the ventricles of the heart empty and contracted, while the auricles were distended with dark blood. The kidneys were not examined.

When I myself, nearly twenty years ago, took single half-ounce and six-drachm doses of chlorate of potassium, I had a sensation of heaviness and dragging in the lumbar region, and increased renal secretion. I did not examine for albumen.

The case of Dr. Fountain, of Davenport, Iowa, occurring at the very same time, is first mentioned by Alfred Stillé (Therap. and Mat. Med., 2d ed., 1874, p. 922). He experimented upon himself, taking an ounce of the chlorate of potassium, and died in a week of nephritis (and enteritis).*

* Alfred Stillé publishes a letter of Dr. John M. Adler, of Davenport, Iowa.

A case of death from chlorate of potassium, occurring in the practice of Dr. Krackowizer, I reported some years ago. It was that of a young lady who was told to use a solution of one ounce of chlorate of potassium as a mouth-wash and gargle. Instead of that, she swallowed the whole of the solution, and within three days died of nephritis.

I have also, before this, referred to one of my own cases; it was that of a man of thirty-odd years, who was told to use internally ten drachms of the chlorate of sodium, within six days. Instead of that, he took the entire quantity

who was Dr. Fountain's friend and medical attendant. According to that letter (Stillé's text makes it an ounce, and I know from my intercourse with Dr. Fountain that he took half an ounce previously), Fountain took half an ounce in a goblet of warm water at 8 A.M., on March 22d, 1861; free diuresis followed during the course of the day; it ceased entirely at 4 P.M. He looked fatigued and was pale, but ate heartily at 7 P.M.; was attacked with purging, vomiting, and cramps after 8 P.M.; was in dangerous collapse at 9.30, with vomiting and purging, with intense pain and cramps, skin cold, with the hue of a person nearly asphyxiated. He rallied, but retained an exceedingly dusky appearance of the skin. Between 6 and 8 A.M. the following day, he voided about two ounces of black-colored urine. After this, there was no secretion from the kidneys. "When he called my attention to the urine, he remarked that he feared the chlorate had seriously injured his kidneys." Immediately after, the choleraic symptoms returned, with profound collapse, but he rallied again; the purging ceased, and there was no further evacuation from the bowels during the six subsequent days of intense suffering. Vomiting and intense pain were incessant. He died just one week from the time of taking the chlorate. "The autopsy revealed a general intense inflammation of the entire alimentary tract, from the stomach to the rectum; portions of the mucous membranes were destroyed, hanging in ragged shreds and patches, as if the intestine had been macerated a long time in a strong alkaline solution. The mucous membrane of the bladder gave a similar appearance. The bladder was empty. There were crystals of the chlorate in the pelvis of the kidneys, and a large bulk of extravasated urine (apparently)" (?) "under the capsule of one kidney." There is no mention made of the anatomical condition of the kidney, but there were two "ounces of bloody urine, and no more for six days," though the patient rallied after that second relapse also. There was general hyperæmia and even ulcerations of the gastro-intestinal tract, whether primary or the result of the pertinacious vomiting and (in the beginning) purging, is another question. It is seen in the violent gastro-intestinal symptoms of nephritis.

within six hours. Within twenty-four hours he suffered from diffuse nephritis. What little urine he passed was smoke-colored, and afterwards black. It contained a large percentage of albumen, blood, hyaline and granular casts. Then there was complete suppression. There was vomiting and diarrhoea, headaches and coma. He died on the fourth day, and the post-mortem examination exhibited acute diffuse nephritis.

Dr. J. Lewis Smith, in a meeting in which the above statements of mine were referred to, reports a case of a child three or four years of age (see *Medical Record*, p. 397, 1878), who took three drachms of the chlorate of potassium in one day. After that only a few drops of bloody urine were discharged, and the child died at the end of twenty-four hours.

In the same number of the same journal Dr. Hall reports a case of a child under one year of age, who took one drachm of the chlorate of potassium in a single night, with exactly the same symptoms and the same results.

Conrad Küster (*D. Zeitsch. f. prakt. Med.*, 1877, No. 33), for no other purpose but to prove the essential identity of the punctated, maculated, membranaceous, croupous, and nephritic forms of diphtheria—similarly to most writers since Bard, Bretonneau, and myself amongst many—reports the following cases:

A young woman of twenty, vigorous and blooming. Mild angina. Small, white specks on tonsils. Feels pretty well. A strong solution of chlorate of potassium for gargling and internal administration. No doses, however, reported. The doctor found her dying at daybreak the following morning. Relatives said that vomiting and diarrhoea commenced in the evening, but that they all slept and were awakened in the morning by the labored breathing of the patient. No post-mortem examination was made; urine was not obtained. There was no dropsy, but the skin exhibited a peculiar dusky hue.

A man of thirty, in vigorous health. Trifling macu-

lated diphtheritic angina. Strong solution of chlorate of potassium as a gargle, and internally lime-water, besides. The tonsils cleared rapidly, but some malaise all the time. Urine albuminous. The doctor learned that the urine was peculiarly black on the third or fourth day. Gradual improvement, but urine albuminous a year and a half after.

A boy of three years, in good health; very mild, punctated, diphtheritic angina. Two other children had diphtheria seriously half a year previously, one of which died of laryngeal diphtheria. Gargle and administration of a strong solution of chlorate of potassium. Next day the doctor was notified the child was dying, and had passed black urine. So it was. The urine *was* black, a little greenish hue, moderately albuminous, the surface bluish white, the child dying. A good deal of vomiting. No dropsy. No post-mortem.

A girl of four, also robust and vigorous. Mild angina, some trifling whitish marks, hardly visible in the tonsils. Gargles and administration of chlorate of potassium in strong solution. Appears nearly well both locally and generally, within two days, but in the afternoon very suddenly: vomiting, yawning, apathy, bluish-white complexion, accelerated and compressible pulse, skin cool. In the evening some urine, black with greenish hue, albuminous, contained hematine. On the following days, the color became more normal, and albumen less. On the fifth day, the danger was over, but the pulse remained frequent a long time. No dropsy. A slight return of albumen on the sixteenth day.

Now Dr. Küster claims all of these cases as acute nephritis, and adds verbatim: "There is here a peculiar resemblance to renal irritation from carbolic acid poisoning. One is reminded of a medicinal poisoning, and would presume its presence if *carbolic acid* had been used for external application. In my cases, the substance irritating the kidneys could be *none but the chlorate of potassium*. How-

ever, as this effect of chlorate of potassium has not been observed, as nephritis in diphtheria is, besides, nothing unusual, the latter must be claimed as the cause of the accidents."

Küster's facts are correct, his theory is not. His cases were mild, all of them tonsillar, no general symptoms, no adenitis; in fact there is no, or very little danger, in consequence of the absence of lymph-vessel communication between the tonsils and the rest of the body. Two of his four cases terminated fatally in a very short time; two barely escaped. The same symptoms, the same nature of the disease in all. The cases seemed to the author like so many of poisoning by medication, *and so they were*. Unfortunately the author, otherwise known as careful, earnest, and conscientious, reports no doses, but in every case he speaks of *strong* solutions of chlorate of potassium, which appear to have been used rather indifferently or indiscriminately. Whoever has followed my remarks, and compares my own cases with his, will not hesitate to look upon his cases as such of acute nephritis brought on by excessive doses of chlorate of potassium.

After all the previous remarks, the practical point I wish to make is this, that chlorate of potassium is by no means an indifferent remedy; that it can prove, and has proved, dangerous and fatal in a number of instances, producing one of the most dangerous diseases—acute nephritis. We are not very careful in regard to the doses of alkalies in general, but in regard to the chlorate we ought to be very particular. The more so as the drug, from its well-known either authentic or alleged effects, has risen, or descended, into the ranks of popular medicines. Chlorate of potassium or sodium is used perhaps more than any other drug I am aware of. Its doses in domestic administration are not weighed but estimated; it is not bought by the drachm or ounce, but the ten or twenty cents worth. It is given indiscriminately to young and old, for days or even weeks, for the public are more given to taking hold of a

remedy than to heed warnings. Besides, it has appeared to me that acute nephritis is a much more frequent occurrence now than it was twenty years ago. Chronic nephritis is certainly met with much oftener than formerly, and I know that many a death certificate ought to bear the inscription of nephritis instead of meningitis, convulsions, or acute pulmonary œdema. Why is that? Partly, assuredly, because for twenty years past diphtheria has given rise to numerous cases of nephritis; partly, however, I am afraid, because of the recklessness with which chlorate of potassium has become a popular remedy.

In this respect the medical profession has done nothing to check its quackish use. For, with the exception of Alfred Stillé, who knew Fountain's case and the destructive powers of chlorate of potassium—it appears, however, that not the kidneys but the intestines were suspected—almost nobody has suspected it. Still, Isambert noticed, after large doses of the drug, increased elimination of urine, and after doses of twenty grammes a sensation of heaviness and pain in the renal region, without any other disorder.

Buchheim (*Arzneimittellehre*, 1859), it is true, speaks of the irritation of the mucous membrane of the urinary tract, with frequent desire to micturate, and even of inflammation of the bladder and hæmaturia resulting from the use of the chlorate, as well as of the nitrate of potassium. Edlefsen, on the other hand, declares the remedy perfectly harmless (*D. Arch. f. klin. Med.*, XIX., p. 97).

In connection with a paper of mine on this subject (*Med. Record*, 1879, March 15th), which he quotes, F. Marchand (*über d. gift. Wirkung des Kali chloricum in grösseren Dosen*, *Sitzgsber. d. Naturforschenden Ges. zu Halle*, Feb. 8th, '79, and *Virch. Arch.*, vol. 77) has also observed intoxication by potassium chlorate. Death ensued either suddenly, or after some days. In the former cases, the blood was brown, but no changes took place in the organs; in the latter, an affection of the kidneys resulted in the emission

of a urine loaded with decomposed blood-cells and depended on obstruction of renal capillaries by the changed blood-corpuscles. Experiments made on dogs, which consisted in potassium chlorate being either administered internally or thrown into the abdominal cavity, had the same effect. Blood taken from time to time proved an increasing discoloration of the same; it resembled the changes taking place in blood after severe burns of the surface. When death did not occur suddenly, the urine changed after twelve or twenty-four hours. Spectroscopic examination of such blood yielded the character of methæmoglobin (the same changes which take place when blood is mixed with potassium chlorate).

Finally, E. Baginski,* referring to my own and Marchand's communications, reports four clinical observations of a nature similar to that of my own cases. I have no doubt but that the number of unfortunate cases of the kind will increase unless my warning be heeded.

My views concerning the treatment of diphtheria are based on the principles laid down in the discussion of the pathology of this disease. Although diphtheria is, after all, a constitutional disorder, yet it frequently has a local inception; in other words, the infection enters into the general system at a certain circumscribed locality. In many cases, this locality is the same, namely, the fauces. From this stand-point, constitutional diphtheria may be compared to the septic absorption occurring in wounds and in the puerperal condition, and for that reason we shall be obliged to rely principally on the method of treatment that is most serviceable in diseases of wounds, and puerperal fever after delivery, to wit: local disinfection.

While, however, we may congratulate ourselves on a positive knowledge of the effects of disinfecting agents upon accessible putrid fluids, we have no proofs of our ability to

* Ueber toxische Wirkungen des Kali chloricum, Arch. f. Kinderheilkunde, 1880, p. 100.

disinfect the blood in the living body. True, we may claim that we possess remedies which enable the body to resist the action of the poison, but we cannot yet assert that we have at our command remedies which, when absorbed into the blood, are able to destroy the poisonous elements contained therein. It is possible that salicylic acid forms an exception; yet concentrated salicylic acid, when mixed with diphtheritic scrapings from the tongue, did not destroy the infectious qualities thereof. Besides, it ought not to be overlooked that salicylic acid, when introduced into the system by internal administration, forms at once salicylates; mainly sodium salicylate, which is not a disinfectant.

Moreover, if the pathological process in question, and pathological processes in general, were the result of bacteria, and bacteria only (or, according to the botanist Naegeli, organized substance of some kind), the therapeutics would necessarily consist in destroying these bacteria, these organized substances. But, when we read of the assurance with which antiseptic medicines are recommended for internal administration, we are certainly entitled to our share of astonishment. It is a well-known fact that the most thorough mixture of antiseptic with putrid material is required to destroy bacteria, but with the most child-like faith do we send our liquids down into other people's stomachs, expecting every antiseptic drop to look out for its duty. It is expected to be absorbed, and being swept into the circulation, to find its way to the "nests" of micrococci stowed away in gangrenous tissue out of reach of vascular or lymphatic circulation, to fall upon the enemies of tissue and mankind and commence their deadly combat. It is also expected that a fraction of a grain of sodium benzoate atomized down into a pulmonary abscess swarming with bacteria should have the same effect; while in the bottle and in the test-tube, we know that a thorough shaking with a well-known and large pro-

portion of antiseptic admixture is required for a satisfactory effect.

N. C. Scharrenbroich's and Appert's (Virch. Arch., 61, p. 364) observations on the effect of quinine are, for the time being, of not much importance for practical medicine. It is true that leukocytes become dark and seemingly granulated in a solution of neutral muriate of quinine, 1 : 200-1 : 2000, and that amœboid movements are imperceptible afterwards. It is also true that doses of $\frac{1}{3000}$ or $\frac{1}{4000}$ of the weight of the body of a frog render both emigration and marginal position of leukocytes impossible, and that the above-mentioned changes will take place; and further, that pulse and circulation are very much retarded. But such doses cannot be employed in practising on the human being, and the full effect of the drug cannot, therefore, be obtained, provided that the doses required in men are in any way proportionate to those in the frog. Even though they be much smaller, like those calculated by Binz, they would be too large to be administered.

Prevention, after all, is but in part the business of the physician. It is mostly that of the individual, or the complex of individuals, viz., town, state, nation. Those sick with diphtheria must be isolated, though the case appear ever so mild, and if possible, the other children sent out of the house altogether. If that be impossible, let them remain outside the house, in open air, as long as feasible, with open bed-room windows during the night, in the most distant part of the house, and let their throats be examined every day. The watching eye of a father or mother will discover deviations from the norm, so that the physician can be notified. Let the temperatures of the well children be taken once a day, in the rectum. Ten minutes of a mother's time are well paid by the discovery of a slight anomaly, which may require the presence of the physician. Happily, there are many mothers who keep and value a self-registering thermometer as an important

addition to their household articles. The attendant upon a case of diphtheria must not get in contact with the rest of the family, particularly the children, for the poison may be carried, though the carrier remain well, or apparently well. Unnecessary petting of the patient on the part of the well ought to be avoided, kissing forbidden, the bed-clothing and linen to be changed often, and disinfected, the air cool and often changed.

The well, or apparently well children of a family that has diphtheria at home, must not go to school nor to church. The former necessity is beginning to be recognized by the authorities and teachers, and also in consequence of partially enforced habit by parents, the latter will be resisted longer. Schools ought to be closed entirely when a number of cases have occurred. Even when the school children have not been affected to a great extent, but a diphtheria epidemic has commenced in earnest, it will be better to close the schools for a time. If that be not advisable, the teacher ought to be taught to examine throats, and directed to examine every child's throat in the morning, and return home every one barely suspicious.

In times of an epidemic, every public place, theatre, ball-room, dining-hall, tavern, ought to be treated like a hospital. Where there is a large conflux of people, there are certainly many who carry the disease with them. Disinfection must be enforced by the authorities in regular intervals. Public vehicles must be treated in the same manner. That it should be so when a case of small-pox has happened to be carried in such, appears quite natural. Hardly a livery stable keeper would be found who would not be anxious to destroy the possibility of infection in any of his coaches. He must learn that diphtheria is, or may be, as dangerous a passenger as variola. And what is valid in the case of a poor hack, is more so in that of railroad cars, whether emigrant or Pullman. They ought to be thoroughly disinfected in times of an

epidemic, in regular intervals, for the high roads of travel have always been those of epidemic diseases, and railroad officers and their families have often been the first victims of the imported scourge. Can that be accomplished? Will not railroad companies resist a plan of regular disinfections because of its expensiveness? Will there not be an outcry against this despotic violation of the rights of the citizen, the independence of the money bag? Certainly there will be. But there was also, when municipal authorities commenced to compel parents to keep their children at home when they had contagious diseases in the family, and when a small-pox patient was arrested because of endangering the passengers in a public vehicle. In such cases, it is not society that tyrannizes the individual; it is the individual that endangers society. And society begins at last, even in America, to believe in the rights of the commonwealth, and not in the rights of the democratic person only. The establishment of State and National Boards of Health proves that the narrow-hearted theories of the strict constructionists have not only disappeared from our politics, but also from the conscience and intellect of society.

The sick-room must be kept cool, the windows kept open—more or less—in the night, the floor frequently washed, the linen soaked at once, the excrements removed. Dead bodies ought to be kept moist, for infectious material, chemical or otherwise, will spread more easily when dry. Attendants must not talk unnecessarily over the mouth or diphtheritic wounds of the patient, and will do well to carry a little dry loose cotton—to be changed often—in each of the nostrils, for it prevents the transport of infection from septic material to such as would be considered exposed under ordinary circumstances (Wernich in F. Cohn's *Beitr.*, III., 1859, p. 115).

A very urgent and important mode of prevention consists in disinfection. Its requirements may be stated as of a double nature. Those who still believe in bacteria as the causes and

main representatives of infectious diseases do not consider disinfection complete unless the disinfectants used have succeeded in destroying the vitality of bacteria. Now, about the same time experiments have been made in both hemispheres, the results of which are singularly unanimous. Thus Schotte and Gärtner, under the orders of the Surgeon-General of the German Navy, publish a report with experiments on the question, how much carbolic acid or how much sulphurous acid is required for the destruction of bacteria ("Zur Tödtung des kleinsten Lebens"),* with the following result, viz., that a sure disinfection cannot be accomplished on board ships by the evaporation of carbolic acid or by burning sulphur, because of the large quantities required for that end, but that there is a fair hope to find a substance which, when in solution, is capable of surely destroying bacteria in watery solutions or in garments. And George M. Sternberg, U. S. A., in his experiments with disinfectants, made at the request of the National Board of Health (Bull. No. 47, May 22d, 1880), comes to the following conclusions, viz., that the amount of pure carbolic acid required to destroy the vitality of bacteria is equal to about seventeen pounds in a room twelve feet square and twelve feet high (capacity 1728 cubic feet) and to fulfil the conditions of the experiments on a large scale it would be necessary to scatter this amount over the floor of a room having these dimensions, and to suspend articles to be disinfected near the floor for at least six hours, care being taken that all apertures are closed, so that the fumes of the acid may not escape. Another experiment (No. 43 in the series) shows that four times this amount (sixty-eight pounds) of "crude" acid placed upon the floor of a room of the same size would not destroy the vitality of bacteria exposed in the room for six hours. These experiments show that the popular idea, shared, perhaps, by some physicians, that an odor

* D. Viertelj. f. öffentl. Gesundheitspflege, 1880, XII., p. 337.

of carbolic acid in the sick-room, or in a foul privy, is evidence that the place is disinfected, is entirely fallacious, and, in fact, that the use of this agent as a powerful disinfectant is impracticable, because of the expense of the pure acid and the enormous quantity required to produce the desired result.

The warning is appropriate, and when it will be heeded, much carelessness will be avoided, and much danger averted. But, perhaps, the case is not so bad, after all, as we might conclude from the results of the observations of the medical men engaged in the above experiments. The uniformity of their results proves almost to a certainty that they are quite correct in regard to bacteria, but those who do not see in bacteria the cause and essence of all infectious diseases, especially diphtheria, will look to disinfection as their safeguard, without fearing to be baffled in their practical efforts to avert disease. Two hundred years ago, when the first infusoria were discovered, they were also accused of being the cause of infectious diseases. They were met with the thunder of cannons; but when that refused to be effective, it was attempted to bewitch the infusoria with music. A pound of pure carbolic acid to a hundred cubic feet looks very much like the cannon ball, but has the advantage, according to Sternberg, of being effective; may be that what the National Board of Health has recommended to us, in the shape of their Circular No. 1,* is also more effective than the music of old. I cannot do better than to copy it from Bulletin No. 10, September 6th, 1879.

Instructions for disinfection.

Disinfection is the destruction of the poisons of infectious and contagious diseases.

Deodorizers, or substances which destroy smells, are

* Signed by George F. Baker, Phila. ; C. F. Chandler, New York ; Henry Draper, New York ; Edward G. Janeway, New York ; Ira Remsen, Baltimore ; S. O. Vander Poel Albany.

not necessarily disinfectants, and disinfectants do not necessarily have an odor.

Disinfection cannot compensate for want of cleanliness nor of ventilation.

I. Disinfectants to be employed.

1. Roll-sulphur (brimstone) for fumigation.

2. Sulphate of iron (copperas) dissolved in water in the proportion of one and a half pounds to the gallon; for soil, sewers, etc.

3. Sulphate of zinc and common salt, dissolved together in water in the proportion of four ounces sulphate and two ounces salt to the gallon; for clothing, bed-linen, etc.

NOTE.—Carbolic acid is not included in the above list for the following reasons: It is very difficult to determine the quality of the commercial article, and the purchaser can never be certain of securing it of proper strength; it is expensive when of good quality, and experience has shown that it must be employed in comparatively large quantities to be of any use; it is liable by its strong odor to give a false sense of security.

II. How to use disinfectants.

1. In the sick-room.—The most available agents are fresh air and cleanliness. The clothing, towels, bed-linen, etc., should, on removal from the patient, and before they are taken from the room, be placed in a pail or tub of the zinc solution, boiling-hot if possible.

All discharges should either be received in vessels containing copperas solution, or, when this is impracticable, should be immediately covered with copperas solution. All vessels used about the patient should be cleansed with the same solution.

Unnecessary furniture—especially that which is stuffed—carpets and hangings, should, when possible, be removed from the room at the outset; otherwise, they should remain for subsequent fumigation and treatment.

2. Fumigation with sulphur is the only practical method for disinfecting the house. For this purpose the rooms to be disinfected must be vacated. Heavy clothing, blankets, bedding, and other articles which cannot be treated with zinc solution, should be opened and exposed

during fumigation, as directed below. Close the rooms as tightly as possible, place the sulphur in iron pans supported upon bricks placed in wash-tubs containing a little water, set it on fire by hot coals or with the aid of a spoonful of alcohol, and allow the room to remain closed for twenty-four hours. For a room about ten feet square, at least two pounds of sulphur should be used; for larger rooms, proportionately increased quantities.

3. Premises.—Cellars, yards, stables, gutters, privies, cesspools, water-closets, drains, sewers, etc., should be frequently and liberally treated with copperas solution. The copperas solution is easily prepared by hanging a basket containing about sixty pounds of copperas in a barrel of water.

4. Body and bed-clothing, etc.—It is best to burn all articles which have been in contact with persons sick with contagious or infectious diseases. Articles too valuable to be destroyed should be treated as follows :

A. Cotton, linen, flannel, blankets, etc., should be treated with the boiling hot zinc solution; introduce piece by piece; secure thorough wetting, and boil for at least half an hour.

B. Heavy woollen clothing, silks, furs, stuffed bed-covers, beds, and other articles which cannot be treated with the zinc solution, should be hung in the room during fumigation, their surfaces thoroughly exposed, and pockets turned inside out. Afterwards, they should be hung in the open air, beaten, and shaken. Pillows, beds, stuffed mattresses, upholstered furniture, etc., should be cut open, the contents spread out and thoroughly fumigated. Carpets are best fumigated on the floor, but should afterwards be removed to the open air and thoroughly beaten.

5. Corpses should be thoroughly washed with a zinc solution of double strength; should then be wrapped in a sheet wet with the zinc solution, and buried at once. Metallic, metal-lined, or air-tight coffins should be used

when possible, certainly when the body is to be transported for any considerable distance.

SPECIAL TREATMENT.

The local remedies may be conveniently divided into three classes. The first includes those which dissolve the pseudo-membrane, and thereby afford an opportunity to remove it; the second, those which appropriately modify the surface from which the membrane has been removed, or the membrane itself; the third, the real antiseptic agents which are credited with being able both to bring about chemical changes and to destroy parasitic organisms, and which are, therefore, believed to be appropriate by those who consider diphtheria either due to a chemical poison or to the presence and rapid proliferation of bacteria.

It is mostly when the pseudo-membrane has its seat in the larynx that it is highly important to dissolve it as rapidly as possible. Of the vast number of remedies that have been recommended for this purpose, but four have held their ground up to the present day, to wit: Lime-water, glycerine, lactic acid, and steam.

INHALATIONS OF STEAM.

Quite remarkable effects have been expected of, and claimed for, them. It is true that pseudo-membranes, like everything else, become softened by the warm vapors. It is also probable that steam increases the secretion of the mucous glands, and thereby possibly loosens the overlying membranes and favors their removal, but it must not be forgotten that it also softens the healthy tissues, and that this change in character enables the poison, whatever be its nature, to penetrate more deeply into them. These two hypotheses must be kept in mind when, in any case, the question of the employment of steam arises.

Steam for the purpose of softening the tissues and of provoking the secretion of mucus and suppuration has

been used to a considerable extent; in fact, in England and America it constitutes an important part of the treatment of diphtheria of the larynx. The patient must inhale it directly from a vessel, or in a tent which is more or less closed, or breathe the atmosphere of the room after it has been saturated therewith. For the latter purpose, water is kept constantly boiling, or lime slaked, or red-hot stones put in water from time to time. The results from this procedure in diphtheria of the larynx have not always been pleasant. I have repeatedly had the joy of seeing children, with croup, become less cyanotic after their removal from an atmosphere of vapor, and I can readily see that pure atmospheric air would be more agreeable and wholesome to a child with stenosis of the larynx than an atmosphere laden with steam. Of course, this remark does not apply to cases of pseudo-croup and bronchitis, which are generally benefited by a warm, moist atmosphere. In pharyngeal diphtheria I anticipate but little from the softening and suppuration-producing properties of steam. Whoever has noticed the obstinacy with which diphtheritic membranes and infiltrations resist all treatment, for days, and even beyond a week, will hardly attribute the recovery from a mild and favorable case of diphtheria of the tonsils, and of light pharyngeal diphtheria, to moist air. Those, however, who deem it judicious to employ steam as a vehicle for carbolic acid, salicylic acid, chloride of sodium, chlorate of potassium, or lime, had best resort to the atomizer for applying these remedies. It can be used without trouble; most children are sufficiently intelligent to allow a spray of nebulized solution to be directed upon the fauces and larynx every ten or fifteen minutes, in case of necessity.

On the other hand—and again I emphasize the fact, that I know of no specifics for diphtheria, and recommend no uniform treatment for all persons, and all cases—I have seen cases of fibrinous bronchitis getting well, when I had every reason to attribute the recovery to the persistent

use of steam. As in the case of croup I have detailed in another place, the child was kept in steam and turpentine vapor more than four days, so I have seen Dr. F. Zinsser lock up a baby in a small bath-room, with one window, and let the hot water run persistently, for days, fill the room so as to produce a constant fog, and make every person in the room dripping. The result was highly gratifying; the baby got well; and so did another, whom I had the good fortune to benefit by my experience in that case. Again, I insist, steam will improve, steam will impair. *Ars longa.* Individualizing is a great art. In regard to the steam therapeutics it is, however, not so difficult. Its object is to soften, but principally to increase the secretion from the mucous membrane, and thereby throw off the superjacent membrane. This can be done to advantage only where there is a natural tendency to it, that is, where there are a great many muciparous follicles under a cylindrical or fimbriated epithelium. This is the condition on part of the pharynx, but not on the tonsils, in a small portion of the larynx, in the trachea and bronchi, not on the vocal cords. Wherever there is pavement epithelium on the normal surface, and where the membrane is imbedded into the tissue, steam can hardly be expected to do good. In the other cases it will. Thus the locality of the diphtheritic process determines to a great extent whether steam is indicated or not. If it be used, the necessity of a full supply of atmospheric air must not be disregarded. Steam, with an over-heated room and without pure air, is liable to be as injurious as steam in pure air is beneficial in a number of cases.

There can be no better proof for the necessity of individualizing, and the impossibility of treating all cases alike, than the fact that many will do well under steam treatment, and others are certainly injured by it. We ought not to be surprised at the repetition of the same old experience that, when two do the same thing, it may not be the same thing. Two means as well patients as doctors.

The object for which steam is inhaled is to soften and remove membranes. When that can be accomplished without reducing the required amount of oxygen, all is well; when, however, respiration is annoyed or interfered with, the contra-indication to steam is as clear as its indication is in more favorable cases. Nor is this different in cases of obstinate pneumonia, where steam may be either beneficial or injurious, according to circumstances.

WATER

may be made serviceable in quite a different manner. Its effect, when taken in large quantities, under normal or abnormal circumstances, on the skin is a matter of daily experience. Copious perspiration is its immediate result. The very same effect is produced on all integuments and amongst the mucous membranes, principally on those of the respiratory and digestive organs. It is particularly plain when water is drunk during a nasal catarrh, when the discharge increases immediately; while, on the other hand, abstinence from drinking reduces the secretion. Much drinking moistens the mucous membranes, rhonchi become looser and moister, and the aim of raising and macerating membranes, if not reached, is certainly rendered more accessible. While one is done, however, the other need not be omitted, and a judicious combination of the methods of supplying the muciparous glands with plenty of fluid suggests itself readily to the thoughtful practitioner in the appropriate cases. Besides professional hydropathists, I know of but one (C. Rauchfuss, in C. Gerhardt's *Handb. d. Kinderkr.*, III., 2, 1878) who favors the plentiful use of water, either by itself, from 100-200 grammes (3 to 6 ounces) every hour, or oftener, or mixed in alcoholic beverages, warm punch, etc.

COLD WATER, AND COLD IN GENERAL,

are useful in different ways. Severe inflammatory symptoms, in diphtheria and other affections, such as redness

of the throat, great pain, swelling of the glands, require cold applications, either an ice-bag or ice-cold cloths, well pressed out and frequently changed. They must, however, be placed where they can do most good; in laryngeal diphtheria around the neck, in pharyngeal diphtheria and glandular swelling over the affected part. Therefore, the flannel cloth which covers the whole of the application must be tied over the head, and not behind. When ice-bags are used, care is to be taken lest they should be too large; if so, they will not affect the desired spot at all. Small pieces of ice frequently swallowed are greatly relished by the patient; water-ices in small quantities will render the same service; ice-cream, in half-teaspoon or teaspoon doses every five or ten minutes, adds to the necessary nutriment. When the fever is high, and the surface hot, sponging with tepid or cold water, or water and alcohol, will mitigate both. For the cold bath or the cold partial pack (trunk and upper part of thighs), the general indications hold good. As a rule, I favor the latter. For many cases have such a tendency to debility and collapse that sometimes the circulation of the surface of the body is badly interlered with by cold bathing. Therefore, a contra-indication to cold bathing must be found at once in cold feet, either before or after a bath. When, unfortunately, the feet do not recover their normal temperature in a very short time, they ought to be warmed artificially, and the cold bath not repeated. In such cases, the cold pack, however, is still indicated. A linen or cotton cloth, large enough to cover the trunk and half of the thighs, is dipped in cold water, well pressed out, and the body of the patient wrapped tightly in it. The arms remain outside, the whole body is then wrapped up in a blanket, the feet may be warmed meanwhile when necessary, and the cold pack repeated as often as required to reduce the temperature, viz., once every five minutes, every half-hour, every hour.

The contra-indications to the use of cold have in part

been alluded to. Very young infants bear it but to a limited extent. The beginning of recovery contra-indicates it, unless for some local cause, for instance, an inflamed gland. Extensive use of cold water or ice is also forbidden when there is no fever, where there is perhaps an abnormally low temperature, where we have to deal with the septic or gangrenous form of diphtheria, where the vitality is low, and the mucous membranes pale or even cyanotic. In such cases, on the contrary, while unlimited internal stimulation is required, the hot bath, or hot pack, and hot injections into the bowels will be found beneficial.

LIME-WATER, GLYCERINE, AND LACTIC ACID

decidedly dissolve the membranes, but whether there is sufficient time in most cases to produce a curative effect is another matter. Concerning lime-water and glycerine, I have employed a combination of equal parts of both. In cases of diphtheria in children of three or four years and over, I think that my favorable results were owing to assiduous cleansing of the throat and nose. In vastly more than one hundred cases after the completion of tracheotomy, I have employed the same combination, reduced to a spray by means of the atomizer, and directed into the opening in the trachea, but must confess that my results left much to be wished for. Particularly in the last few years, in which the prevailing epidemic of diphtheria hardly ever intermitted, my results from tracheotomy have been very unsatisfactory, because, amongst other measures undertaken for the same purpose, the spray of lime-water and glycerine was not of the least service in preventing the descent of the process into the bronchi. Long ago I have begun to rely less implicitly upon lime-water, where a local action upon the larynx and trachea is called for, owing to its instantaneous conversion into carbonate of lime.*

* Dr. Billington takes exception to my remarks on the inefficiency of lime-water made before the New York Academy of Medicine, on the occasion of

LACTIC ACID, too, dissolved in from ten to twenty-five parts of water, has yielded no better results in my hands. I can cite but one case, that of a boy of five years, who, under the constant spray of lactic acid into the throat, and as far as possible into the air-passages, recovered from an attack of croup after a number of days, although tracheotomy had not been performed. A similar success has been communicated to me by Dr. Wm. Chamberlain. Those cases of tracheotomy which I subsequently treated with a spray of lactic acid did not terminate more favorably than those in which lime-water and glycerine were employed. I have not been able to convince myself of the locally solvent action of PEPSIN. With NEURIN I have no experience. It was both externally and internally first used by Winiwarter, and is recommended because of its being an alkaline antifermentative, while all other

the doctor's reading a detailed account of forty cases of diphtheria. In an open letter to the Editor of the Medical Record, over his name and address, he says it occurred to him that the question might be answered by a very simple experiment. He says: "I was permitted, by a patient who has an unusually patent and tolerant throat, to hold a bit of red litmus paper at the end of a wire, and protected from the action of the saliva by coils of wire, well back in the pharynx, the patient being instructed to breathe naturally. I then, with the atomizer which I use in treating diphtheria, and in exactly the same manner, threw the spray of lime-water into the throat. In fifteen seconds the red litmus paper was turned blue—this change occurring quite as rapidly and as completely as when the same experiment is performed in the outer atmosphere. In other words, it was not appreciably modified by the breath. Again, litmus paper moistened with lime-water can be held in the breath for some minutes before its blueness is perceptibly affected by it. This experiment seems to me to show that lime-water spray reaches the fauces and pharynx as lime-water, and does not immediately thereafter cease to be lime-water. When spray thrown into the throat by this method enters the larynx, it does so by being drawn in thither by the inspired breath which is comparatively free from carbonic acid, and it therefore reaches the walls of the larynx as lime-water, and then continues to be lime-water for a certain length of time."

So the red litmus paper, while dry, was not easily affected by the carbonic acid of the breath? Why should it, when no chemical text-book makes a statement to the contrary? And the red litmus paper was turned blue by covering it with lime-water—indeed! Litmus paper owes its very existence

antiseptics are acid, and, therefore, must undergo changes before they can be admitted to the circulation. It is claimed that the foetid odor disappears readily, and that membranes are dissolved more easily by its three-per-cent solution than other dissolvents.

C. Edel recommended (*Med. Rec.*, Jan. 19th, 1878) the treatment of diphtheria by

TURPENTINE INHALATIONS.

Fifteen drops of oil of turpentine are inhaled from a common inhalation apparatus, which is placed at a distance of three inches from the mouth of the patient, for a period of ten minutes every hour. He claims recoveries in from twelve to forty-eight hours. Taube (*Deutsche Z. f. prakt. Med.*, 1878, No. 36) also uses oil of turpentine, but, as far

and reputation to the fact that it is so very sensitive, and shows the effect of either acid or alkali so readily, and in the most minute quantities. Let us remember that the atomizing apparatus with each pressure sprays about 1-500th of a grain of lime; that, therefore, it takes hours to send a grain of lime into the throat; that, however, a membrane has to lie immersed in lime-water for hours before it shows signs of maceration; that lime-water introduced into the trachea even, through the tracheotomy tube, does not dissolve membranes to any satisfactory degree—though they be only deposited upon, and not (as on the tonsils and parts of the larynx) into the tissue; and though in the trachea the mucus of thousands of glands readily aids in macerating; and though in the opened trachea the lime-water certainly reaches the membrane, which is not so certain in regard to pharyngeal or even laryngeal membranes when you atomize into the mouth and pharynx. And as the doctor relies on experiments, he can add one which he made when he was quite young. Let him blow into as much as half a pint of lime-water (containing about four grains of lime), and the whole of that lime will be a turbid cloud of carbonate after a very few expirations.

Here is another experiment: Let the doctor dip blue litmus paper into moistened carbonate of lime, or a solution of sodium carbonate, or even bicarbonate, and his blue litmus paper will behave exactly as it does in his patient's throat, to wit, it will remain blue; which is not remarkable at all; and then let him breathe upon the litmus paper moistened with calcium carbonate ever so long, and it will remain blue. Should it not? In his experiments on his patient's throat nothing is proved except that the newly formed calcium carbonate secures the blue color to his litmus paper for some time. That is all, and requires no proof.

as the effect of the latter is concerned, it cannot be appreciated, as his treatment is not a uniform one. For he adds two or three daily injections into the tonsils of a three-per-cent solution of carbolic acid, one or two teaspoons of claret every hour, ice externally, two or three warm baths daily, with cold shower, milk, egg, infus. digital. 0.5:80.0, with ac. benzoic 1.0. etc. His dose of oil of turpentine is like that of Edel; in order to avoid local irritation, he oils the face and covers the eyes with a bandage; he reports that he never saw renal or cerebral disorders following its administration.

For years I was in the habit of using turpentine, either the oil or the rectified spirits, as an inhalation in bad cases of pneumonia, where hepatization was very extensive, and expectoration and resolution did not commence, with very good result in children and adults. The vapors of turpentine are so volatile and penetrating (and certainly the procedure of Taube so disagreeable to the patient, if it be permitted at all by children) that the usual method of inhaling from an apparatus appeared to me to be very superfluous. I allow the patient to remain in his bed, and keep water boiling *constantly* on an alcohol lamp, on the stove, or over the gas. A tablespoonful of spir. rect. or ol. terebinth., more or less, is poured on the water, care being taken that nothing is spilled in the fire. Thus the room is constantly filled with a penetrating odor of turpentine, which is not at all disagreeable, even when in great concentration. The effects are very satisfactory indeed. Where circumstances allowed or required it, I raised a tent over the bed, large enough not to give inconvenience to the patient, and to admit either the whole apparatus or the tube containing the mixed vapor of water and turpentine. This plan I followed also in the case of laryngeal diphtheria of a girl of two years, in the children's service of the Mount Sinai Hospital. The baby was in a room of her own, with a nurse. A tent was raised over the bed. Four days and nights was she exposed to the water and

turpentine treatment, awake or asleep ; not only she, but also the nurse, whose presence under the tent was insisted upon by the patient whenever she was awake. It ought to be stated that the case was not (or was not allowed to become?) a very serious one. It was serious enough to be diagnosticable, to produce hoarseness, aphonia, dyspnoea, and to render the perception of pulmonary murmurs impossible ; but there never was cyanosis with the exception of a slight hue on the upper lip. She got well, with no other treatment but my iron and pot. chlor. solution. As a practical addition, I may say that the nurse did not suffer much more than she would have done after the same time passed in a close room, and in constant attendance upon an exacting and whimsical patient.

AMMONIUM CHLORIDE,

muriate of ammonia, may sometimes be used to advantage for its softening and liquefying effects. Its internal administration in bronchial and tracheo-laryngeal catarrh is so old that it has several times been obsolete. Of late, more stimulant effects have been attributed to it than it actually possesses. But its liquefying action, in cases where the secretion of mucus is defective, and expectoration slow and viscid, is undoubted. Thus it proves valuable in many cases of simple catarrh, both when administered internally and inhaled. The latter mode of inhalation I have often resorted to, and believe that its macerating influence has been of service to me in cases of laryngeal diphtheria. Half a teaspoonful of the pure salt is spread on the stove, or burned over alcohol or gas. It evaporates immediately and fills the room, or the tent, with a white cloud, which, when dense, excites coughing. But it does not irritate to any uncomfortable degree, and the process may be repeated in an interval of an hour or more.

HYDRARGYRUM

has been used in all and any children's diseases. When I

was young, I seldom saw a prescription for a child without some little or much mercury on it. That, therefore, it should be given in diphtheria is not surprising; that, however, it should be given in a septic or gangrenous form is almost incredible; that it should be recommended as a panacea in all classes and forms of diphtheria shows that common sense and sound judgment does not always prevail in the treatment of a disease where individualizing is of the utmost importance. If there be any specific in the world, mercury is not it; not even in syphilis. However, not all cases of diphtheria are septic or gangrenous; and not all cases occur during an epidemic; nor are all the cases occurring during an epidemic of the same type. Some have the well-pronounced character of a local disease, either on the tonsils or in the larynx. The cases of sporadic croup met with in the intervals between epidemics yield no constitutional symptoms, and assume more the nature of an active inflammatory disease, very much like the sporadic cases of fibrinous tracheo-bronchitis. These are the cases in which mercury deserves to have friends, apologists, and even eulogists. Amongst them I shall not mention any of the old-time practitioners, who may have been led to the regular use of mercurial preparations in large doses by the very fact of their cases assuming the merely inflammatory character, but a few with a deservedly fair fame, and taking a high rank in the modern profession. I shall mention Oppolzer, Bartels, Senator, and Rauchfuss, the two latter of whom are alive and still active; but again insist on the fact that they use mercurials in membranous croup for its liquefying and melting effect. Of these, however, after long experience, Bartels discarded it; Oppolzer used calomel and iodide of potassium; Senator, calomel and antimony; Rauchfuss, calomel with oxysulphuret of antimony, blue ointment, and hypodermic injections of the corrosive chloride.

In regard to the action of mercurial remedies, I am no longer so skeptic as I was a quarter of a century ago. For

a dozen years I hardly ever prescribed mercury, supposing that the harm it might do could be avoided by substituting other medicines, and that its effect, except in syphilis, could be obtained by other means. I admit that the experience of many subsequent years has changed my views to a certain extent. I know that in chronic inflammatory troubles which I considered incurable in former times, a good many favorable results have been due, at my hands, to the protracted influence of mercurials; thus, for instance, in chronic inflammations of the nervous centres, particularly the spinal cord. I also know that when the constitutional effect of mercury could be obtained speedily, cases of fibrinous tracheo-bronchitis got well in an unexpected manner. To accomplish that, it is necessary to give small doses very frequently. Calomel, 0.5-0.75 (grs. viij.-xij.), divided into thirty or forty doses, of which one is taken every half-hour, is apt to yield a constitutional effect very soon. Such doses, with minute doses, a milligramme or more (gr. $\frac{1}{80}$), of tartar emetic, or ten or twenty times that amount of oxysulphuret of antimony, have served me well in fibrinous tracheo-bronchitis. But the mucous membrane of the trachea and bronchi is more apt to submit to such liquefying and macerating treatment than the vocal cords. The latter have no muciparous glands like the former, in which they are very copious. And while the tracheal membrane is apt to be thrown out of a tracheal incision at once, though of more recent date, the pseudo-membrane of the vocal cords takes from six days to sixteen or more for complete removal. Still, a certain effect may even here be accomplished, for maceration does not depend only on the normal mucus of the muciparous glands, but on the total secretion of the surface, which will be in constant contact with the whole respiratory tract. Thus, either on theoretical principles, or on the ground of actual experience, men of learning and judgment have used mercury in such cases as I detailed above, with a certain confi-

dence. The actual benefit derived therefrom cannot have been great, for the mortality from croup has nowhere been encouraging. Nor is it an enjoyable proof of its efficacy that Bartels is known to have lost confidence in it in his ripest old age, either for its general unsatisfactoriness, or for the reason that the general character of all the cases in the epidemics of his later years changed the nature of his cases from the inflammatory to the septic type.

If ever mercury is expected to do any good in these cases of suffocation by membrane, it must be made to act promptly. That is what the blue ointment does not. In its place I recommend the oleate, of which ten or twelve drops may be rubbed into the skin, along the inside of the forearms or thighs (or anywhere, when their surface becomes irritated) every hour or two hours. Or refracted doses will be useful, such as given above; or hypodermic injections of corrosive bichloride in one-half (or one) per cent solution in distilled water, four or five drops from four to six times a day, or more, either by itself or in combination with the extensive use of the oleate, or calomel internally. The hypodermic injections act very promptly and favorably, as I repeatedly convinced myself; for instance, in those cases of hereditary syphilis, which, from the presence of volar or palmar pemphigus and general cutaneous eruptions at birth, yield, as a rule, an almost fatal prognosis under ordinary circumstances, and with the ordinary treatment.

ASTRINGENTS.

It seems to me a fact worthy of notice that the pure astringents, as alum, tannin, and nitrate of silver, which are so extensively employed in the treatment of the simple pharyngeal catarrh, appear to have been given up, to a greater or less extent, by most practitioners, where an exudative process exists. Oertel raises an objection to the employment of astringents, on the ground that, by

hindering the detachment of the membranes, they are more likely to prevent suppuration, and thereby facilitate the impregnation of the tissues with poisonous elements. Whether this theory be correct or not, I assume that it is based on an experience which agrees with my own, as a result of which I am opposed to the employment of pure astringents. The remedy, however, which, for a number of years, has been employed by physicians, and which, furthermore, belongs to the group of astringents, is the

CHLORIDE OF IRON.

This substance was first used in diphtheria because an analogy was supposed to exist between the latter disease and erysipelas, in which the remedy had proved of great utility. It was introduced into France by Velpeau, into England in 1851 by Hamilton Bell in the treatment of erysipelas, and employed, not merely locally, but also internally. It has been used in diphtheria in France, by Gigot, since 1848, and in the following year by Crichton, in Scotland. Richardson, in the year 1865, published a report of two hundred and twenty cases of diphtheria in Melbourne, extending over a period of seven years, which he had treated without exception, since 1861, with large doses of the chloride of iron in combination with chlorate of potassium administered in the form of powder. There were eighteen deaths, constituting 8.2 per cent of the entire number of cases, in other words, a mortality but little greater than that from measles, as observed by himself and many others. Since that time, British and American journals have teemed with the reports of good results following the administration of the chloride of iron, and in a monograph which appeared a few years ago, Schaller maintains that the diluted chloride of iron is superior to all other remedies in the treatment of diphtheria. I have used the chloride of iron in very many cases of diphtheria. In my essay on the latter disease, in 1860, I spoke of its effectiveness after observing a large number of cases of the disease in 1858, 1859, and 1860.

In the administration of the chloride of iron it must by no means be forgotten that small doses at long intervals are out of the question. I have not the least doubt but that the failure of the remedy may be attributed in most cases to the fact that the doses were too small and administered too seldom. Steiner thought himself in duty bound to refute Schaller's statement concerning the efficacy of the remedy after employing it for four children. He administered hourly a teaspoonful of a mixture containing five to eight drops of the tincture of the chloride of iron in three ounces. In addition, a mixture containing thirty drops in sixty grammes was applied locally three or four times daily. The youngest two, one a child of three years, died in consequence of an extension of the process to the larynx, the other two recovered.

These experiments were decidedly incomplete and therefore gave an unsatisfactory result. A dose of five to fifteen drops every ten or fifteen minutes, half hour, or hour, is indispensable for a proper estimation of its effects. Gargles are not of much service, for the simple reason that they do not come into sufficient contact with the affected parts, and reach at the utmost to the anterior pillars of the soft palate. A direct application of the remedy to the mucous membrane of the pharynx may also be desisted from, thereby avoiding any irritation, the internal administration at short intervals causing the pharynx to be sufficiently influenced by local contact with the remedy. It must, of course, not be expected that the chloride will remove the membrane, but it can frequently be seen to reduce the hyperæmia and swelling, and prevent the reproduction of exuded material. Now, as regards the power of coagulating albuminous substances which is possessed by astringents, a considerable difference between these various agents can be demonstrated. A solution of tannin brings about a cloud-like flocculus, carbolic acid the same, but it requires to accomplish that effect about half a minute; alum, a viscid coagulated mass which

sinks to the bottom of the test-tube without increase of its bulk by further deposits; creasote, added drop by drop, forms isolated coagula, each one of which sinks separately to the bottom and increases in size. Perchloride of iron produces a coagulum several lines in thickness and sinks slowly to the bottom, while the fluid above remains clear (M. Putnam Jacobi).

When we expose the subcutaneous veins in a living rabbit and touch them with a drop of solution of the chloride, no effects will be apparent for more than a minute. At the end of that time, the calibre of the veins becomes decidedly diminished; on the other hand, a drop of creasote similarly applied gives rise to coagulation which at once obliterates the veins by compression. The effect in the latter case is purely mechanical. The former indicates that the chloride exerts a decided influence on the vital contractility of the blood-vessels. This increased contractility certainly assists in diminishing the rapidity of absorption of putrid fluids through the blood-vessels, which constitutes the principal source of danger from the disease.

It cannot yet be positively asserted that the chloride of iron exerts a direct effect on the lymphatic vessels. Naturally this was claimed, when the remedy was transferred from its therapeutic effects in erysipelas with the accompanying inflammation of the lymphatic vessels of the skin, to the treatment of diphtheria. Although we know of no direct compression of the lymphatic vessels due to the action of the chloride, yet it may be assumed that perhaps the compression of the blood-vessels exerts a similar influence upon the neighboring lymphatics. In consequence of this there would be an impediment to the absorption and further development of poisonous substances in the lymph. The chloride, like the sulphate of iron, is a tolerably powerful disinfecting agent. All astringents act more or less as disinfectants, and some of the best disinfecting agents, as creasote, are powerful astringents.

gents. Mundy employed iron in the treatment of wounds, and Beale claims that it is a powerful antiseptic, especially when combined with glycerine. He explains its action on the ground of its limiting the growth of the bioplasm and hence checking the rapid necrobiosis. It is as efficacious in checking the movements of micrococci and bacteria as in coagulating albuminous ferments, so that its action may be explained to suit the existing theories concerning diphtheria. As the result of experiments with disinfecting agents, which were undertaken in London, it was found that 2.27 litres (half a gallon) of chloride of iron sufficed to disinfect 30,000 litres (6,600 gallons) of polluted water, 1.36 kilogramme (3 lbs. avoirdupois) of chloride of lime, and 36.35 lbs. of lime being necessary to produce the same result. The chloride of iron long had a place in Chevallier's list of disinfectants, and is placed by Herbert Baker by the side of other mineral salts to which he attributes the power of destroying chemical poisons.

The internal administration of the chloride of iron, moreover, is undoubtedly as important as its local application, even though the theory of its absorption, and of its action after absorption, be still involved in obscurity. It has been claimed that the chloride is decomposed immediately after it has been introduced, and that the muriatic acid alone enters the circulation, but the urine has just as little revealed the presence of free muriatic acid as of the salt. There is reason to believe that the chloride of iron is absorbed with remarkable rapidity by the stomach directly, and that the re-appearance of the iron in the fæces may be explained by an elimination of the remedy by the intestinal glands. Moreover, Quincke has found that when the chloride of iron is rapidly introduced directly into the veins of animals, emboli form in the pulmonary vessels; but when the drug is so slowly injected that its entrance into the blood may correspond with the period required by the stomach for absorption, only very minute precipitates will form and be taken up by the white blood-

corpuscles. If this observation be correct, it may go very far toward explaining the action of the chloride of iron in septic diseases, which are accompanied by an exalted activity of the lymphatic vessels, and an increase of the white blood-corpuscles. Furthermore, Saase has in so far modified the general opinion concerning the influence of the iron and the obliteration of the blood that he attributes to the ferrous salts the power of converting oxygen into ozone. They share this power with the blood-globules exclusively, and could hence, to a certain degree, supply a deficiency of the latter. Pokrowsky, too, has shown that iron increases the process of oxydation in the body by demonstrating that in health there is a elevation of temperature and an increase of the percentage of urea in the urine, during its administration. In anæmic persons, to whom iron has been given for the purpose of increasing the amount of blood, the above phenomena may be observed before this object is accomplished. Thus iron appears to replace the blood-corpuscles to a certain extent. Now, in infectious disorders of the blood, when the red globules are perpetually menaced with destruction, it seems plausible that the preparations of iron should exert an antiseptic action.

Finally it has been found that of all the preparations of iron, the chloride possessed the power of stimulating the nervous system. Possibly this effect may be traced to an increase of the arterial pressure in the nerve-centres. It has been said that this effect has been vividly illustrated in certain forms of chlorosis. If this be true, iron would be all the more indicated in diphtheria, since it would act as a prophylactic against a series of nervous phenomena that so frequently present themselves, both during and subsequently to, the diphtheritic process.

CARBOLIC ACID

has long held a prominent position in the group of disinfecting agents. It is an established fact that, in solutions

of the proper strength, it checks putrefaction, destroys bacteria, and suspends the movements of the white blood-globules. It is true we cannot infer from this that diphtheria depends on the presence of living germs which, in a state of health, exist in large numbers in almost all the organs of the body, the more so as an immense quantity of the disinfectant is shown above to be required for that purpose; but carbolic acid exerts a powerful influence on the vitality of all living elements, and, hence, too, on rapidly proliferating epithelium which constitutes a part of the diphtheritic membrane. It has been experimentally proven that carbolic acid destroys the efficacy of vaccine virus; in a similar manner, probably, it lays low the diphtheritic poison. I employ it both locally and internally, the latter in frequently repeated doses—every ten or fifteen minutes to one hour—dissolved in water, with or without the addition of glycerine or alcohol, administering from one-half to two grammes (eight grains to half a drachm) in twenty-four hours. For gargles, mouth-washes, and nasal injections, I resort to solutions of one-half to two per cent. Rothe also has seen excellent results from the use of carbolic acid, and it forms an important part of many recipes highly recommended by contributors to medical journals. In the degree of dilution in which he has employed it, the results have probably been similar to mine with the use of more concentrated solutions, for he too describes the contraction and shrinking, though not the crumbling of the membrane which I have frequently seen to occur in a short space of time. He combines the remedy with iodine, for external applications, in the following proportion: Carbolic acid and alcohol, each 2 parts; water, 10; tincture of iodine, 1 part.

SALICYLIC ACID,

of late, has been highly praised as a disinfecting agent. Its action is tolerably well understood, but continued observation and clinical experience will tend to cool

the ardent enthusiasm with which salicylic acid has been praised. I have not had favorable results from the local employment of salicylic acid. In rather concentrated solutions (1 : 30-50) and in weaker strength (1 : 200-300) its action was alike undeserving of praise. In the more concentrated form it acts as a caustic; the only effect that I could perceive from the milder solution was a diminution or total disappearance of the foul odor from nose and throat, but I cannot testify to a more rapid detachment of membrane, or to a more speedy termination of the disease under its use. Its failure to produce good results seems to me to be in direct proportion to the extent and thickness of the membrane. Its salts are no disinfectants, but antifebriles, and salts are at once formed in the stomach when it is given internally. Where there was high fever accompanied by a very moderate exudation, I had reason to be satisfied with the effects of the drug administered internally. I have reference to cases in which the general symptoms are more prominent than the local ones, where the latter may even be absent, and to which, as long as twenty years ago, I applied the term of diphtheritic fever. One of my first cases on which I tried the salicylate of sodium was that of a boy of four years, who for days had a slight exudative deposit, a marked swelling of the glands of the neck, and a temperature of nearly 106° F., without showing any signs of improvement. The prognosis was rather unfavorable, or, to place it in the best light, very doubtful. Under the administration of 4-5 grammes (3-4 scruples) of salicylic acid, combined with 3 grammes ($2\frac{1}{2}$ scruples) of bicarbonate of sodium daily, the boy recovered. My experience was similar in many other cases. In many, on suspending the remedy, the temperature would rise, but sank again as soon as it was resumed. For this reason I recommend the use of sodium salicylate as an antifebrile agent in a severe attack of diphtheritic fever, while I am not at liberty to speak favorably of the local action of salicylic

acid on parts covered with membranous deposits.* Whenever it is administered, however, it ought not to be forgotten that serious brain troubles, collapse, and irregular and paralytic breathing may follow its administration. It ought not to be used without careful watching, and the cotemporaneous free use of alcoholic stimulants.

As regards the antiseptic action of the usual doses of

QUININE,

it can be hardly considered as brought about otherwise than by actual contact with the membrane, and not perchance after absorption into the blood. Binz found, as the result of experiments with solutions of pure quinine varying from one part in a hundred to one in a thousand, that the latter sufficed to prevent the development of bacteria in fluids capable of undergoing putrefaction; but even estimated thus, a patient with eighteen pounds of blood would require one hundred and thirty-eight grains of quinine circulating therein in order to satisfy the conditions of Binz's experiment. If Binz considers two grammes (half a drachm) of quinine per day sufficient for an individual weighing one hundred and twenty pounds, his calculation is founded on experiments with dogs, in which

* In regard to the effects of sodium salicylate, P. A. Blanchier made a number of experiments in Vulpian's laboratory (*Recherches expér. sur l'action physiol. du salicylate de soude*, Paris, 1879). His conclusions are as follows: It requires rather large doses to exhibit an effect. In the commencement it stimulates, and afterwards paralyzes the central nervous system to such a degree as to destroy the functions of the gray substance. By its effect on the nervous centres, and especially the medulla oblongata, it increases secretions, produces vomiting, and disorders the gastro-intestinal tract, and destroys life by paralyzing respiration and circulation. It has no influence on the peripheral, sensitive, or motory nerves, but paralyzes the sympathetic ganglionic cells in general, and the intracardial ones particularly. In very large doses it affects the glandular cells as well, as also, histochemically, the muscular tissue, though it cannot claim to be a muscle poison. Its therapeutical success in articular rheumatism is attributed to its local modification of the inflamed tissues of the joints, an attempt rather at an explanation than a lucid and intelligible illustration of its effect.

septicæmia was avoided by the injection of quinine. It is also necessary to bear in mind that Binz makes a distinction with regard to the preparations of quinine employed. He warns against the use of the bisulphate as being the most inactive. No matter which preparations are used—I prefer the muriate—I have come to look upon quinia as of no great service in reducing the temperature in infectious fevers. The main indication for its use can only be found in inflammatory fevers. When it is given, however, salicylate of sodium may be added for a short time to obtain a speedier effect.

My position in regard to the question whether the local manifestation of the disease, or local origin of the disease, should be treated with caustics, is at once determined by the fact whether in individual cases I consider the membrane the symptom of a general disorder, or the cause of the disease. In the former case less can be accomplished than in the latter, supposing that the destruction of the morbid products can be accomplished at all. The matter is not by any means simple, for even though the membrane be but the result of a general process, yet the presence of membrane or of an infiltration acts in turn locally, by influencing the lymphatic system, by injuring the blood-vessels, and by contaminating the air, so that, what was an effect now becomes a cause. Therefore there is, at all events, a theoretical indication to destroy existing membranes, and thus render them innocuous. But in practice we often meet with either impossibilities or contra-indications. Most of the caustics act only superficially; this is especially the case with NITRATE OF SILVER.

POTASSA

and other deliquescent salts, as also

CHROMIC ACID

may at once be excluded because of their proving dan-

gerous to the neighborhood. In my experience, CONCENTRATED MINERAL ACIDS penetrated no deeper than nitrate of silver; for the quantity at each application must only be small. Indeed it is far easier to recommend than to carry out the cauterizing process. Few patients have enough self-control to permit a thorough application of the remedy, and rarely does it succeed so happily that a satisfactory effect is obtained, while at the same time—and that is of paramount importance—no injury is caused to the surrounding parts. Inasmuch as I so forcibly insist on the importance of keeping the oral mucous membrane as healthy as possible, and even for that reason alone look with favor upon the treatment by chlorate of potassium or sodium, I should certainly take great care not to cause wounds or erosions on which the diphtheritic process could at once take root. I consider it wrong to cauterize a membrane or infiltration unless I am sure of being able to do it thoroughly, and at the same time to limit the action of the remedy to the diseased surface. I prefer, above all other remedies for cauterization, a mixture of equal parts of carbolic acid and glycerine or the concentrated acid alone. I have occasionally seen good results therefrom. The local action is at all events satisfactory if it can be restricted. The membrane crumbles and drops off in small fragments. Where the oral or pharyngeal space is small and the patient unruly, I confine myself to frequent disinfection of the diseased parts with weak solutions of carbolic acid, by way of the mouth or nose. I never use force to compel a child to submit to a cauterizing process in the throat, when I mean to limit the effect of a caustic. Dr. A. Hadden recommends to me the local application of the liquor subsulphatis ferri. He states that in his opinion some of his tracheotomized patients recovered in consequence of the local effect it had on the tracheal membranes.

My experience with

BROMINE,

administered internally at short intervals, in order to

combine local with general treatment, and employed in numerous cases in several epidemics, does not redound to its credit. To apply bromine in substance, or slightly diluted, to the affected parts, as I have seen others do, and have myself done in hospital gangrene, is here entirely out of the question. Therefore, solutions only are admissible, as recommended by Ozanam, Schütz, Rapp, and others. I have given the remedy in one-half per cent solution with bromide of potassium every half-hour or oftener, without being edified regarding the local or general effects. In a number of cases, I have preferably given the bromide *in statu nascente* by mixing chlorine water with a solution of bromide of potassium. It is a more agreeable mode of administering the remedy, although the effects are not intensified thereby.

In this connection, however, I should feel remiss of fulfilling my whole duty if I did not refer to the favorable effects, claimed by as deserved a colleague as Prof. Wm. H. Thompson, for bromine both locally and internally. While in a number of cases I have carefully followed his plan of treatment, I cannot say that my expectations were fulfilled. Still, his convictions are so strong, and his field of observation so large, that I gladly avail myself of his permission to detail his treatment in his own words, as contained in the following letter of his:

NEW YORK, Aug. 21st, 1880.

Prof. A. Jacobi:

DEAR DOCTOR:— . . . I will simply give my reasons for relying on bromine in diphtheria as follows:

I have been led long ago, by experience as I fancied, to prefer the haloid disinfectants, *i. e.*, chlorine, bromine, iodine, and sulphur, to the carbolic acid class, *i. e.*, quinine, salicin, carbolic acid, camphor, the spices, etc., against the septic changes which the specific communicable acute diseases cause in the system. On the other hand, for infection by decomposed pus, etc., the carbolic acid group is superior to the haloids. Those conditions, however,

which are more or less similar in their symptomatology, with low petechial or gangrenous manifestations, common in bad cases of measles, scarlatina, variola, typhus, and some cases of dysentery, as well as markedly in diphtheria; in all such I regard the chlorine and bromine class far more effective.

Of all members of this latter group of antiseptics, I have found bromine the most active and the best borne when administered according to the directions to be mentioned.

It can be taken internally in relatively larger doses than any disinfectant with which I am acquainted, and from the entire absence of effect upon the nervous system (which cannot be predicated of any of the carbolic acid family, not even of quinine) it acts on the body purely as an antiseptic. Hence I have employed it for eighteen years in the treatment of diphtheria to the exclusion of all other remedies, and until I note very different results from it than has been the case in my own practice, I shall use no other remedy whatever.

The benefits I would claim to result from its proper administration in diphtheria are these:

1. When applied locally, it promptly arrests fetor by arresting directly the gangrenous process, and thus lessens risk from absorption.
2. It acts as an anti-putrefactive likewise in the fluids of the body generally, *i. e.*, blood, interstitial circulation, and secretions, owing to its high rate of diffusibility, equal to sodium chloride itself.
3. It locally destroys the communicable property of the discharges, shown by the immunity of attendants from any sore throat, when it is used, and from its checking the spread of the disease in the locality.

No claim can be adduced for it as an antidote, so-called, to the diphtheritic agent, except that I believe it can wholly destroy its germs when they are locally developed previous to general infection. When reproduction throughout the body has occurred, or when the contiguous

lymphatic glands are extensively infiltrated, its action is then simply that of an internal antiseptic, and as such, I think, has no superior.

When called to a case, I order two solutions to be used; the first of equal parts of Lawrence Smith's solutio bromini and glycerine, applied with a hair pencil to the membrane, as gently as possible. Sometimes I use the solution full strength. The brush should be washed at once in water, and does not last more than one day, owing to the action of the bromine on hair. If, however, the membrane be very extensive and the parts much swollen, or difficult to reach, I resort instead to douching with a Davidson syringe, using half a drachm to one drachm of the solution to the pint of warm water. By beginning gently with the stream directed against the buccal mucous membrane, the child soon becomes accustomed to the current and allows it then to play against the deeper parts.

Internally I order from six to twelve drops of the solution to half-ounce of sweetened water, every hour, two, or three hours according to the urgency of the case, and continuously; no other medicine being taken until the disappearance of the membrane; when the case may then be treated on general principles. For convenience' sake I frequently order the preparation: \mathcal{R} Smith's sol., 3 i.; Aq., \mathfrak{z} i.; teaspoon in tablespoon of well sweetened water. It should be swallowed promptly, for the disagreeableness of bromine is due much more to its fumes than to its taste, and patients soon learn to take it readily. The only inconvenience which I have seen from it has been in some a slight looseness of the bowels, which may be readily controlled by a small dose of paregoric. It is well borne by the stomach, as I have repeatedly seen it retained when solutions of quinine, or tr. ferri were uniformly rejected. The only diet recommended is cold milk and lime-water.

As to the amount which may be taken with impunity, I once saw, in consultation with Dr. Chauveau, of Houston street, an infant of fourteen months, whose case seemed

hopeless from laryngeal extension of the membrane, but for three days it took twelve drops of Smith's solution, equivalent to two drops and a half of pure bromine, every hour unintermittingly, and finally recovered without an untoward symptom.

The most convenient way of making Smith's solution is: take two ounces of a saturated solution of potass. bromid. in water, add to this, very slowly, in a bottle and with constant shaking, one ounce of bromine. It is better to add a part and then let it stand a while before adding the rest; then fill up gradually, and with constant shaking, with water till it measures four ounces. This solution should be complete and without sediment. Dose, as above stated, six to twelve drops in well sweetened water. But it should not be ordered in a mixture with either glycerin or sugar, for it is soon changed by these agents into a colorless compound which is certainly inefficacious. For daily use I order it as above stated, dissolved in different strengths in simple water, and if not exposed to too strong light, it keeps for several days. Yours sincerely,

W. H. THOMSON.

Finally, I reproduce (from *Prag. Med. Woch.*, No. 10, 1880) an abstract of the treatment which J. Schütz recommends. It is a bromine treatment, similar to that which he eulogized ten years ago, and which, amended by what I cannot help believing doubtful practice, he applied to twenty-eight cases with, as he states, satisfactory results.

As soon as a deposit is visible in the throat, the finger is covered with a piece of linen cloth, moistened with water, and the membrane rubbed off.* When it is but partly removed, the process is repeated. "Patients felt better immediately, were cheerful, fever diminished, appetite increased, and sleep was no longer disturbed." After

* Also E. J. Bonsdorff (*Hygeia*, No. 4, 1879, *Med. Rec.*, Sept. 20th) takes the responsibility of mechanical removal, by all means possible, of the exudation and mortified tissue, and touches the surface with nitrate of silver, twice daily "if necessary."

the membranes were rubbed off, he made two or three injections of a solution of bromine and potass. bromid., ãã 1, in 200 parts of water, or of pure water. That forcible removal of membranes must be repeated two to three times daily. The injections are to be repeated hourly. "Advanced children take a great pleasure in making the injections themselves." (?) "The parts injured during the rubbing-off process remain mostly intact." (?) When there are obstinate membranes, they ought to be moistened with bromine solution five or six times daily. Cold applications round the neck are not required. Glandular swellings require pot. iodid. ointment (1:4), the size of a pea, three times daily. (?) "The patient is isolated. As a matter of precaution, after recovery, the bedding is aired, and it is left to the attendant to resort to any kind of disinfection." (!)

Jaeger (Corr.-Bl. f. Schweizer Aerzte, 1877, No. 5) uses iodide of bromine, potass. brom., ãã 0.3-0.5; aq. destil., 150.0 (gr. v.-viiij. in water $\frac{2}{3}$ v.), $\frac{1}{2}$ -1 teasp. hourly or half-hourly for inhalation. Ice in addition. Netolitzky (Prag. Med. Woch., 1879, June 23d), potass. brom., bromin., ãã 0.05-1.0; 150-200 (gr. i.-iss. in $\frac{2}{3}$ v.-vi.) to be inhaled from a sponge for five or ten minutes every hour. Prince (St. Louis M. J., 1877, July 18th) prefers, also for inhalation, iodin., 0.06; potass. iod., 0.25 (gr. i.-iv.), in a mild solution.

OZONE

has but recently been recommended again, this time by Ph. Jochheim (Darmstadt, 1880). It is developed in a Richardson's apparatus, with two tightly-fitting corks and a funnel, by slowly (drop by drop) adding concentrated chemically pure sulphuric acid, 30.0 ($\frac{2}{3}$ i.), to potassium permanganate, 30.0. Ozone is developed while permanganic acid and manganese hyperoxyde are formed, and expelled by a common syringe balloon, the tube of which enters the apparatus by the perforated cork. Potassium iodide paper has to turn purple or bluish-black by it. An

inhalation of three or five minutes every hour or two is considered sufficient.

Still he does not neglect other treatment. Locally he uses a two or three per cent solution of potassium hypermanganate as a gargle, mouth-wash, or application; internally quinia muriate, 0.03-0.12 (gr. ss.-ij.), in honey or wafer, every two hours. Steam is used besides, and as a disinfectant, potass. hypermang., 50.0, in water, 300.0 (1:6), mixed slowly in a china vessel with pure concentrated sulphuric acid, slowly heated.

BORIC ACID

has been used and eulogized by Wertheimer as a gargle, in a solution of 10 : 300 or 250, which is to be used every hour, with the exception of the nights, where the intervals may become longer. If gargling be inconvenient or resisted, the solution is injected, or the nasal douche resorted to. M. Vogel (*Allg. Med. Centr. Z.*, Nos. 99 and 100, 1876), brushes the throat and gargles with ac. bor. 4-6 : 180, every hour in the commencement.

My own experience is not gratifying; it is true that I gave up the remedy after I had used it in a dozen cases, but my results did not appear to encourage me.

J. T. Lewis (*Brit. Med. Jour.*, Jan. 11th, 1879) recommends sulphurous acid in water as a gargle, in combination with plenty of plain food and also stimulants.

Concentrated solutions of boric acid have been highly praised in diphtheritic conjunctivitis. It is to be applied hourly.

In accordance with Graham's experiments, made in Prof. Klebs' laboratory, which appeared to show that a certain proportion of

SODIUM BENZOATE

prevented the vegetation of the diphtheritic contagion, L. Letzerich (*Berl. klin. Woch.*, Feb. 17th, 1879) administered sodium benzoate in twenty-eight cases, part of which

were serious. Of these twenty-eight, but one died, and that was a case of laryngeal diphtheria. The child had suffered from croup before, and retained a great tendency to laryngeal disorders. Infants of a year or under, took one-half tablespoonful hourly of the following mixture :

℞ Sod. benz.....	5.0 (℥iv.)
Aq. destill.,	
Aq. menth. pip.....	āā 40.0 (℥i. 3 iij.)
Syr. cort. aur.....	10.0 (3 iiss.)

Children of from 1-3 years took 7.0-8.0 daily (3 ij.) children of 3-7 years, 8.0-10.0 (3 iiss); and those over 7, sod. benz. 10.0-15.0 (3 iij.-℥ ss.). Adults took 15.0-25.0 (℥ ss.-3 vi.) daily. A disagreeable effect was not noticed. Twice or three times daily, or in bad cases every three hours, sodium benzoate was thrown or blown upon the diphtheritic deposits. Older children would also gargle with a solution of 10.0 : 200.0. In all cases the fever decreased within twenty-four or thirty-six hours.

Amongst the first who tried sodium benzoate extensively was Demme (Annual Rep., 1878). His doses were, for the age of 3-6 months, 2.5 daily (℥ij.); 7-12 months, 5.0 (℥iv.); 1-2 years, 7.5 (3 ij.); 3-7 years, 12.0-15.0 (3 iij.-℥ ss.). In no case did he see an injurious effect. Beside the internal administration, he blew the drug into the throat every two or four hours. When the process was a very rapid one, with large tumefaction of the neck and glands, he also made subcutaneous injections of sodium benzoate (1 : 2) into the subcutaneous tissue and also into the tonsils. Besides, the trunk was treated with cold packs, and sometimes cold bathing was resorted to when the temperature was above 102°; in septic cases also alcohol 5.0-75.0 (℥iv.-℥ iiss.) daily. His mortality of twenty-two per cent he claims as favorable, though it does not impress me as such. To lose six cases out of twenty-seven is a result no practitioner is apt to rejoice over. Still he insists upon the following points as reliable conclusions: 1st. That sodium benzoate is a reliable antizy-

motie in both internal and external administration. 2d. By its local application both as a powder and in solution it increases the secretion of the mucous membranes and favors the removal of diphtheritic deposits. 3d. Even large doses do not reduce the temperature to any great extent. 4th. The contraction of the heart becomes more intense, the beats less frequent, and the secretion of urine more copious. 5th. It does not influence either nephritis or albuminuria.

In regard to its effects as a medicinal agent, I never expected much. The parasitic school of pathologists have been remarkably hasty in their literary productions, as is well known. The journals of the last ten years are flooded with superficial observations, insufficient experiments, and immature conclusions. "Preliminary communications" of any length, and long articles, at the close of which the very writer says that his experiments prove nothing (compare, for instance, Miflet in F. Cohn's Beitr., III., 1879), abound. Thus journalism, and mainly in regard to those branches which boast of being exact, has become rather flimsy and flighty. The worst feature of this tendency in medical literature consists in the fact that the men who have to rely on their senses mostly, in their special investigations—mostly microscopical—finally rely on their own senses only. When they see benzoate of sodium destroying bacteria in a glass vessel, they not only take it for granted that bacteria are the infectious disease, but also that the human organism will permit of the same action on the part of the antiseptic medicine as the glass vessel. Thus benzoate of sodium is sent into the stomach, or into a pulmonary cavity, under orders to do the same thing it does in the laboratory. The drug has, in consequence, had a short life, after having been extolled in a very limited time by microscopists, Russian diplomats, and the public in general. Clinicians tried it, but have soon learned not to trust it much. I believed I saw some favorable result in puerperal diseases, at first, but do not feel

convinced in regard to them at all. As an anti-diphtheritic, or even as an anti-febrile remedy, it cannot be trusted.

Fr. Mosler's general principles in regard to the treatment of diphtheria are those of all sound practitioners. Thus he avoids depletion, antiphlogistics, and emetics, particularly antimony, and prescribes nutritive food and medicines. As a local application he employs oil of turpentine in inhalation, after having given up carbolic and salicylic acids, and potass. hypermanganate, because of bronchial irritation resulting from their use. After he obtained a favorable result from the use of

OLEUM EUCALYPTI E FOLII

(not *ol. eucalypti australis*, which is lower in price) in a case of *echinococcus* of the lungs, he also employed it in diphtheria. His strongest formula is as follows:

℞ <i>Ol. eucal. e fol.</i>	5.0 (℥iv.)
<i>Spir. vini rectific.</i>	25.0 (℥vi.)
<i>Aq. destill.</i>	170.0 (℥vi.)

M. For ten inhalations, one every hour or one and one-half hours.

The professor hopes that no bronchial irritation will result from these inhalations, but admits that it may. His own cases are not numerous, and his confidence is not great.

As in every disease which, at least in certain instances, presents great and insurmountable difficulties, so too in diphtheria, the pharmacopœia has been ransacked for remedies. Long before the time of Roger and Barbosa,

SULPHUR

had been used. The insufflation of the drug at first gave rise to coughing and vomiting, and in the end proved disagreeable and futile. Still, Stuart (*Practit.*, April, 1879) recommends it again.

THE BALSAMICA,

copaiba and *cubeb*, have been recommended for internal

administration, mainly by the French. Trideau's treatment of the inflammatory stage (not the septic) of diphtheria is so formidable that he was sure to have successors, if not successes. His doses of cubeb powder are for a child of from eight months to a year, 8.0 (3 ij.) daily; of from two to three years, 10.0-15.0 (3 iiss.-3 ss.); for an adult, 25.0-30.0 (3 vi.-3 i.). Roger and Bastian opposed its administration because of its dangerousness, and mainly because of the difficulty of taking or giving it, and of the certainty of disordering digestion, which deserves of the greatest consideration wherever recovery is, as frequently in diphtheria, depending on the power to resist the debilitating influence of the disease. Sanné opposes the use of balsamics, because of their being liable to produce nephritis, temporary though it may be. M. Laruc (*Gaz. Hop.*, 1877, No. 112) followed Trideau, however, adding quinia to obviate or relieve fever. Vedrini (*Gaz. Méd.*, 1878, July 27th, Aug. 3d) saw "great relief to children over ten years, in serious cases even," when he gave cubeb (12.0 [3 iij.] daily), and lost six out of ten. Others have given it, and what is worse still, recommended it. The objection to it, that it deranges digestion, is a very valid one, indeed, and Sanné is not correct when he believes the nephritis following the inordinate use of balsamics to be but temporary. Smaller doses than those ordered above are well known to produce permanent nephritis; unfortunately the opportunity to observe such cases is not so uncommon where diseases of the genito-urinary organs, in which the drugs of that class are so often used, are of frequent occurrence. Vaslin gave cubeb in 20.0 gramme (3 v.) doses daily in mild "anginæ," and in thirty-four cases of croup. Of his eight cases of diphtheritic pharyngitis, one died of paralysis. Of the thirty-four cases of croup, three recovered without, ten with tracheotomy. This is high praise for tracheotomy, none for cubeb.

T. M. Lownds (*L. Lancet*, 1879, March 22d) recommends

tr. perchlor. ferr., 3 iij.-iv.; sol. ac. ammon., 3 iss.-ij.; pot. chlor., 3 i.-iss.; aq., f. 3 viij., tablespoonful every hour.

D. de Berdt Hovell (L. Lancet, Dec. 28th, 1878) commences his treatment with a dose of calomel.

A. Erichsen (Petersb. Woch., 1877, No. 4), hydrargyr. cyan., 0.0006 (gr. $\frac{1}{160}$) to children under three years, 0.0012 (gr. $\frac{1}{80}$) over three years, every hour; every two hours during the night. He did not succeed in losing more than 3 children out of 25.

Collin (Rev. théér., 1876), no cauterization, no depletion, but good nutriments. Aq. calc., 120.0-360.0 (3 iv.-xij.); liq. ferri chlor., 20.0-40.0 (3 v.-x.); ac. carbol., 0.06-0.12 (gr. i.-ij.).

Anthony (Med. Surg. Rep., 1877, Jan. 13th), sod. sulph. carbol., 3 ij. to a child of seven years.

M. G. Sloan reported to the Iowa State Med. Soc., Jan. 27th, 1880 (Med. Rec., Feb. 21st, 1880), 34 cases of diphtheria treated successfully with quinine in large doses, alternated with sulpho-carbolate of sodium. He used locally tinct. ferri. chlor. and glycerine, applied gently to the affected parts, and in four cases of laryngeal complications he cured three by the use of inhalations of lime-water with the constitutional remedies named.

D. McFalls (Med. Rec., Jan. 24th, 1880), Lugol's solution thickened with tannic acid to the consistency of thin cream, as a local application.

Peyrot (Gaz. hebdom., Oct. 17th, 1879), bromide pot., 15-20 (3 ss.-3 v.); aq., 100 (3 iij.), as a local application, also brom. pot., undissolved for the same purpose.

Hagenbach (Child's Hosp. at Bâle, 16th ann. rep., 1878), ice internally, and externally inhalation of aq. calc., a solution of sod. salicyl., lactic acid, or pot. chlor. A separate room filled with steam, zinc chlor. upon the tracheotomy wound. Emetics but rarely.

T. Kaatzer (Berl. klin. Woch., 1877, No. 46) recommends still cauterization with the solid stick on three consecutive days, and pot. chlor. 10:300, a tablespoonful every

hour, gargling with the same solution, and ice externally.

Fehr (*Deutsch. Z. f. prakt. Med.*, 1877, No. 25) gargles with sod. chlor. (on the meat-pickling principle) and carbon. sod. 1 : 150, a tablespoonful every hour, for the reason that, according to Tiegel, bacteria and micrococci are destroyed more easily by sod. carb. than other salts.

H. Beyer (*Brit. Med. Jour.*, 1878, May 4th), inhalation of a lactic acid solution.

H. Roger and Peter (*Un. Méd.*, No. 100, 1877), in mild cases emetics (!), ipec., 0.2-0.8 (gr. iii.-xii.); syr. ipec., 30.0 (℥ i.), teaspoonful every 5 minutes, brushing of the mouth with lemon juice, twice daily irrigation with borax, alum, or aq. calcis. In serious cases repeated emetics (!!), syringing with aq. calc. 4-8 times daily, brushing with caustic soda 25 : glyc. 100, or arg. nitr. 10 : 30 water.

Bartels: ice, pot. chlorate, insufflation of alum or tannin, steam inhalations.

Eidam: inhalations of whatsoever kind, if but warm and moist, warm poultices, pot. chlorate gargles.

H. Zeroni (*Memorab.*, No. 4, 1879), depletion and poultices.

Kingford (*L. Lanc.*, No. 17, 1879), for more than twenty years, liq. ferr. mur., pot. chlorate, glyc. and water, every 2 or 3 hours, two daily applications of tr. ferri and glycerine. Food and stimulants.

A. Schuster, no specific remedy, no antiphlog., but robor., stimul., and symptomatic treatment. Quinia, ether, brandy, camphor, pot. chlor., aq. calc., ice.

John H. Gilman (*Med. Rec.*, Sept. 20th, 1879), locally once or twice daily ac. carbol., gtt. xv.; tr. ferri chlor., 3 iv.; aq., 3 iv. Internally, hourly through the day, pot. chlor., 3 iss.; aq., 3 iv.; ac. mur., gtt. x. A teaspoonful. In worse cases, pot. chlor., 3 iss.; aq., 3 iv.; tr. ferri chlor., 3 ss.-i.; quin., gr. ij.-v., teaspoonful every hour, spray or gargle of liq. sod. chlorin.

v. Rokitansky (*Allg. M. Cent. Z., Med. Rec.*, July 12th,

1879), chloral and water, āā, brush every half-hour. Milder solution when the membr. has disappeared.

F. L. Hartmann (Med. Rec., Jan. 11th, 1879), chlorine in solution, sustaining diet, gargles of pot. chlorate, alum, salicylic acid, syr. ferri iod.

F. A. Hubbard (Med. Rec., Nov. 15th, 1879), sod. hypsulph. and pot. chlor., āā ʒ ss.; may be dissolved in water as required for gargle, spray, internal use. Tully's powder, quinia, brandy, hot fomentations, or camphorated oil over swollen glands. In laryngeal diphth., vapor of lime in hot water from a coffee pot.

M. J. Gahan (Med. Rec., Jan. 18th, 1879), tr. ferri mur., ʒ i.; pot. chlor., ʒ ij.; aq., ʒ vij.; gargle 4 or 5 times a day; tr. ferri mur., 10–20 drops every two hours. Thus in 200 cases, "it has yet to fail me—*when*" the disease was taken at its onset.

E. Wiss (Deutsche Z. f. prakt. Med., No. 34, 1878), sulphate quin., 0.4–0.6 (gr. vi.–x.); aq. destill., 90.0 (ʒ iij.); ac. muriat. dil., gtt. iij.; ammon. mur., 6.0 (ʒ iss.); syr. cort. aurant., 30.0 (ʒ i.); $\frac{1}{2}$ –1 teaspoonful every two hours. No death (any patients?).

Bouffé, no pot. chlor., no alkali, no emetics for external use. Every two hours ointment of axung, 75; camph., 25; tr. benz., 4–8. For internal use every hour a teaspoonful or one-half tablespoonful of: lemon juice, 300; sod. chlorid., sod. sulphate, āā 10; honey, 15; with the addition of some sod. carbonate. In the intervals, flax-seed tea, gargling, milk, touch the lips often with the mixture, keep the neck and chest warm, do not cauterize, ice, soups and bouillon. In albuminuria, milk with or without soda.

Wm. A. Reiter (A Monograph on the Treatment of Diphtheria, Philadelphia, 1878), calomel and ^apot. Somebody is credited on page 29 with "Pittsburgh children are hard to kill."

J. Dubrisay (Gaz. Hôp., 1877) still writes against the extensive use of tart. emetic.

H. Helmkampff (D. Z. f. pr. Med., No. 37, 1877), ice

internally and externally ; after three or four days, inhalation of steam, disinfection of oral cavity by mild solutions of carbolic acid ; robórants. For swelling of submaxillary region a two-per-cent solution of carbolic acid hypodermically.

Taube (*Jahrb. f. Kinderh.*, XIV., 1879, p. 209) : injection into the tonsils of a three-per-cent solution of carbolic acid, frequent injection into the nose, and sprays. Borax is preferred to turpentine, after this had been eulogized a year before. Local application in the night also. At 103°, quinia, a warm bath three times a day with cold shower. Frictions with alcohol. Cold applications around neck every half-hour. Cold pack every hour. No cauterizing. Milk and wine. Windows open. No draught.

MECHANICAL REMOVAL OF THE MEMBRANES

is not permissible unless they are almost entirely detached. As a rule, it is best to wait quietly till they are completely detached and cast off or swallowed, unless partly loosened membranes in the larynx or trachea afford an indication for an emetic. F. Barker alone has been fortunate enough to be able to claim that, with the assiduous and exclusive employment of turpeth mineral as an emetic, he has never had a death from croup of the larynx. Forceps requires very delicate manipulation, as any scratching or eroding of the neighboring mucous membrane increases at once the area of diphtheritic deposits ; sponges and brushes merely remove some superficial coating of the membrane without detaching the latter. Even where the membrane has been thrown off spontaneously, a new one often appears in a few hours. After a mould of the trachea and its bifurcation had been cast off, in a certain case—death occurring seven hours later—an autopsy revealed the presence of a thick membrane at the seat of bifurcation. Furthermore, the reports of good results from attempts at removal of local deposits are occasionally to be received with great care, particularly when they have reference to the larynx.

Undoubtedly camel's-hair brushes and sponges may be thrust down, and in fact membranes removed by them, but in very small quantity compared with that which still remains. At all events, I should not expect good results from such practice in cases of membranous deposits in the larynx. Perhaps still less here than elsewhere. For after tracheotomy, the tube can never be removed before at least a week has elapsed after the performance of tracheotomy; I have never succeeded in doing without it before the seventeenth day in my own cases. Besides, the examination of membranes in the cadaver demonstrates that they have nowhere a more tenacious hold than in the larynx. What success then can be hoped for from attempts at a mechanical removal from that locality? The difficulty I have sometimes met with in my attempts at partly clearing the nasal passages by mechanical efforts makes me hesitate to put much faith in a mechanical clearing out of the larynx.

THE TONSILS.

In mild cases of diphtheria of the tonsils I at times endeavor to destroy the membrane, but only when it can be reached with ease. In my opinion, the indiscriminate use of mineral acids and lunar caustic have done more harm than good. Where I can easily reach the membranes, I usually apply concentrated carbolic acid; where the membranes are not entirely within reach, I desist from this procedure. A scratching of the mucous membrane and a wounding of the epithelium would assist in spreading the membranous process in a very short time to the surrounding parts. I have already discussed the tendency of the disease to extend rapidly, and the danger of creating fresh wounds. In most cases of simple tonsillar diphtheria, I administer small doses of chlorate of potassium or sodium in water, or the tincture of the chloride of iron, so that from two to eight grammes (3 ss.-ij.) are taken in a day. I add a little glycerine, partly for the sake of keeping the

remedial agent longer in contact with the diseased surface, partly for its own antifermentative effects, and give it in short intervals. The accompanying fever is usually not high, and the neighboring glands are as a rule but little swollen or not at all. When there is a slight

SWELLING OF THE LYMPHATIC GLANDS,

cold water or ice applications are usually all that is needed. The latter should be made according to general indications. The glandular (and peri-glandular) swellings are less the result of an actual filling-up with foreign matter than of secondary irritation. Ice has a happy effect in such cases, both on internal administration, in the form of frequent small quantities of ice-water, ice-pills, ice-cream, and iced medicaments, as also externally by ice-cold cloths, or india-rubber bags filled with ice.

In general, the treatment of the swelled glands must be both based on its causes, and adapted to the present condition. The adenitis and periadenitis is of secondary nature, the irritation being in the mouth, pharynx, and nares. In these localities it is where the main treatment is required. The sooner the primary affection is removed, or relieved, or rendered innocuous, the better it is for the secondary complaint. Frequent doses of chlorate of potassium, or sodium, or biborate of sodium (or benzoate?) in mild doses frequently repeated, according to the principles laid down in another part of this book, mouth washes, gargles, nasal injections with water, salt water, or solutions of disinfecting substances are not only indicated, but highly successful. When the case is recent, cold applications are required, but no washes. When it is of older date, stimulant embrocations are in order. Iodine ointments are absorbed but slowly; mercurial plasters do good in some cases; iodide of potassium dissolved in glycerine (1 : 3-4), frequently applied, iodine in oleic acid (1 : 8-12), iodoform in collodion or flexible collodion (1 : 12-15) applied twice daily, the latter frequently with

very good result, are beneficial. Copious suppuration is very rare. Cases in which a free incision meets with an abscess ready to heal are very uncommon. But local abscesses in large numbers, with gangrenous walls and pus mixed with a sero-sanguinolent or sero-purulent liquid are more frequently found. In such cases, a probe introduced into the lancet wound enters easily into the broken-down tissue in every direction, on slight pressure, to a distance of three to six centimetres (several inches), according to the size of the tumefaction. I have seen fatal hemorrhages from such gangrenous destructions; therefore, the treatment must be both timely and energetic. The incision must not be delayed too long. When the skin assumes a purplish hue, or is simply discolored, it is time to incise, and apply concentrated or nearly concentrated carbolic acid to the interior unless the neighborhood of very important blood-vessels or nerves yields a contra-indication to concentrated applications. In that case, a milder preparation is advisable, but the application should be repeated often, until the suppuration becomes more normal. Then mild disinfectant injections into what has now become a cavity will be found satisfactory, particularly when meanwhile the general condition of the patient has been improved.

DIPHTHERIA OF THE NOSE

results either from an extension of the morbid process from the pharynx, or occurs primarily. It occasionally manifests itself by a peculiar, thin, flocculent discharge, not necessarily copious, and at times even trifling, and very often by a very early swelling of the glands of the neck, especially those behind and beneath the angle of the jaw. Nasal diphtheria often occurs where the nasal mucous membrane has for a long time been the seat of catarrh. Especially during the prevalence of an epidemic of diphtheria must we be careful not to allow a nasal catarrh to have its own way; we must likewise guard against con-

sidering the thin and flocculent discharge in infected cases as a mucous secretion. Whatever be the origin of nasal diphtheria, whether primary or the result of a similar affection in the throat, local treatment should at once be instituted, and if this be done, the great majority of cases will terminate favorably. The danger in this form of disease consists in an excessive absorption of putrid substances, and in the breathing of contaminated air. The indications for treatment are clear and decisive. The interior of the nasal cavities must be thoroughly cleaned and disinfected. If this be commenced early, the original seat of the affection may be reached, and the disinfectant process will, as a rule, have good results. It is not necessary to select very energetic disinfectants; a solution of twelve to twenty-five centigrammes (two to four grains) of carbolic acid in thirty grammes (an ounce) of water is at once mild and effective, and hardly gives rise to more discomfort than luke-warm water. Nasal injections must be made very frequently, until each time the stream of fluid has a free exit through the other nostril or through the mouth. They must be made at least every hour, and even oftener if necessary; at the same time it is advisable to be careful that the fluid does not enter the Eustachian tube. This can be prevented, to a certain extent, by compelling the patient to keep the mouth open during the procedure. I have seldom seen evil, or only disagreeable results from the administration of nasal injections in diphtheria. Still, a medical friend assures me that he has seen convulsions to follow an injection, an occurrence I never met with. It is likely that the mucous membrane of the pharynx is swollen as far as the openings of the Eustachian tubes, to such a degree as to render the entrance of fluids into the latter improbable. The hardness of hearing, which is of so frequent occurrence in the course of a severe catarrh or diphtheritic attack, seems to indicate that the mucous membrane of that part is in a state of swelling. An or-

dinary syringe will suffice. However, when administered by parents or nurses, the blunt nozzle of an ear syringe or nasal douche is preferable ; furthermore, by using the latter, the distribution of fluid is more equal. Occasionally here, as in local applications to the mouth and pharynx, the atomizer may be used to advantage ; but the tube must be properly introduced into the nostrils. There are cases of nasal diphtheria, however, which are far more troublesome to manage than the foregoing would seem to indicate. I have seen cases in which the nasal cavities, from the anterior to the posterior nares, were filled and completely occluded by a dense solid membranous mass. I was then compelled to bore a passage with a silver probe, to gradually introduce a larger-sized one, and then to apply the pure carbolic acid, in order to remove the densest and thickest masses, and finally was able to make injections ; even in such cases I have often had the gratification of being able to give a favorable prognosis. The dangerous secondary swellings of the glands will often subside after a steady employment of disinfectant injections for from twelve to twenty-four hours, but it must not be forgotten that these injections require to be made very frequently, either every hour or half-hour. We must not be drawn from our line of duty by the patient's desire for rest and sleep, but must continue the treatment uninterruptedly. It will be found that the children frequently do not object to this method of treatment ; I have even met with some who, after convincing themselves of the relief afforded thereby, asked for an injection. When we are about to bring each injection to a close, it is well to press together the nasal cavities for an instant with the fingers. By this procedure the fluid (unless doing so spontaneously) is forced backwards to the pharynx, and is swallowed or ejected through the mouth and thus washes the pharynx and mouth at the same time. Frequently, however, this latter object is obtained with every injection ; for, the palate being swelled, œdematous, and paretic, the fluid is not

prevented from reaching the pharynx, even in the average case. In regard to the choice of a disinfecting agent, I have but a few words to say. I believe that no one of them has important qualifications above the others. I avoid those which stain, and produce firm coagula. For the latter reason I do not use the sub-sulphate and perchloride of iron; for the former, the permanganate of potassium. I employ, as a rule, carbolic acid in solution, of the strength above mentioned. Where there is but a slightly fœtid odor, I have frequently employed lime-water, or water, with glycerine, or a solution (1:100, 1:50) of chloride of sodium, or sod. bicarb., also sod. biborate. Disinfecting agents and antiseptics, whether carbolic acid, salicylic acid, or iron, are of no service when administered internally only, unless the seat and cause of the septic infection be attended to previously. I refer to what I have said above in relation to iron and salicylic acid. Under the local employment of antiseptics, as described, or by simply washing out with water, or salt water, most cases recover; without them, death will result. This much my experience has assured me of, that there is a certain number of cases which terminate fatally; but it is likewise true that the mortality need not be excessively great. It is a great satisfaction to me learn from a recent paper of R. J. Nunn (*The Indep. Pract.*, Sept., 1880) that my method is appreciated and valued to its full extent. The author speaks very highly of the local treatment with iodine and boracic acid. I cannot grant that it is hard to carry out the exact and apparently barbarous treatment necessary for a favorable result, for it is certainly more barbarous to sacrifice than to save life.

It is a positive fact that when children suffering from nasal diphtheria, with its peculiarly septic character, are permitted to sleep much—and they are apt to be drowsy under the influence of the poison—they will certainly die. To allow them to sleep is to allow them to die.

The first symptom of improvement is often a rapid

diminution of the glandular swelling. But not in all cases of nasal diphtheria these glandular swellings will be so prominent; in fact, it would be expecting too much to suppose that all at once there should be a rule allowing of no exception. The exceptions are of twofold nature: 1st. There is very little absorption through the lymphatic ducts, and very little, if any, glandular swelling in such cases where the very beginning of the disease is marked by slight hemorrhages, or by a discharge of bloody serum from the nostrils. In these cases, the blood-vessels are so superficial that they rupture and aid in macerating and sweeping off the membrane before absorption into the lymph circulation can take place. These cases are not always, however, mild in character. Open blood-vessels do not only discharge, they are also apt to absorb; and thus it is that many of these cases, be the glandular swelling ever so slight, prove very serious, and thus also, that they can be saved by very frequent disinfection only. The second exception is formed by those cases in which nasal diphtheria, or any other, attacks a mucous membrane which has been the seat of chronic catarrh and intestinal cellulitis, with consecutive thickening, induration, and shrinking. In color, thickness, and consistency, a normal tonsil, pharynx, or Schneiderian membrane differs greatly from those which have undergone a hyperplastic tissue-change. In the latter condition, blood-vessels and lymph ducts are compressed and atrophied, and no longer a high road into the system. It is, therefore, rather hazardous on the part of as careful a practitioner as Dr. Ripley (*Med. Rec.*, July 24th, 1880, p. 90) to declare it a folly to expect to cure the disease by any local application; or of trying to prevent auto-infection in a system already charged with the poison. For as there are cases in which its feverless character and the local changes clearly mark a case as probably of merely local origin, local treatment, if it could or can be applied, is indicated in just these cases; and secondly, the "system being charged with the poison"

does not mean an unalterable condition; for while elimination is going on constantly, absorption of new poison is keeping pace with it more or less. Not even death is an unchangeable condition, much less a morbid process. Besides, Dr. Ripley says: "Even on the theory that these children die of septicæmia, and that the poison is absorbed from the nasal cavities, is syringing out these cavities several times an hour indicated? Who thinks of washing out an infected uterus, or abscess of the pleural cavity, or other organ, with any such frequency?" Certainly nobody, but nobody ever thinks of an equality of condition in nasal diphtheria on one hand, and a puerperal uterus or an abscess on the other. Even in the impossible case that all the membranes were washed away by a nasal injection, it is nothing new that the membranes will form again and again, and thus there is always, in addition to the former infection, a new one, and a necessity to meet it. If the doctor says: "If carried out as recommended, it must prove a most exhausting plan of treatment," I refer him to what he relies on, viz., clinical observation, and very much desire he should try and be satisfied.

THE LARYNX.

The severest form of diphtheria is that located in the larynx, "membranous croup." Its pathology has been discussed elsewhere. Its general treatment, whether the disease has originated primarily in the larynx or trachea, or been communicated from the pharynx, does not differ from that laid down for diphtheria in general. Naturally the larynx, with its principal symptom of stenosis (croup) of the organ, viz., the suffocatory phenomena, call for special treatment. This is represented by the administration of an emetic to fulfil the indication of removing mucus or partly detached membranes from the larynx. Such is their only indication in my experience. I never could satisfactorily explain the reason why Fordyce Barker's cases of membranous croup should all get well with no other

treatment except repeated emetic doses of the yellow sulphate of mercury (turpeth mineral). Somewhat like him, Lissdorf (*Memor.*, 1876, p. 263) claims to have lost but five per cent of his two hundred croup cases treated with repeated doses of sulphate of copper. Similar results are claimed by M. H. de Bey (*Beitrag zur Casuistik*, etc., 1879), but he adds: "It is true the diagnosis, in the majority of cases, was made from the well-known sounds of respiration and cough only; in some, however, by the expectoration of membranes." Thus, evidently, the majority of cases were not those of membranous croup, but of "pseudo-croup," or laryngeal catarrh.

The selection of the emetic, when indicated, is of great importance. Antimonials ought to be avoided because of their depressing and purgative effect. Ipecac is but rarely effective. The sulphates of zinc, and particularly of copper, deserve preference. Turpeth mineral acts promptly and satisfactorily. When no emesis can be obtained, the prognosis is decidedly bad.

The mechanical treatment of membranous croup by the introduction of tubes ("tubage") into the diseased larynx has first been recommended by Loiseau, and afterwards by Bouchut. The latter author's enthusiastic praises of that method have contributed more than its deficient success to its speedy downfall. For not only did he claim complete comfort and relief from dyspnoea for the procedure, but instant restoration of the voice. Of late, Schroetter, Weinlechner, and Monti have employed catheters to provide an artificial aperture and dilatation.

In regard to tracheotomy, that last resort in croup, I cannot refrain from stating that, in proportion to the increasing severity of the diphtheritic epidemics, the results of tracheotomy in my hands and in those of others have grown worse and worse. Of sixty-seven tracheotomies which I published twelve years ago, twenty per cent recovered; about two hundred tracheotomies performed by me since that time, brought down

the percentage of recoveries to such a low figure that only the utter impossibility of witnessing a child's dying from asphyxia has goaded me on to the performance of tracheotomy. I here add that I do not wish it to be inferred that I have changed my views concerning the indications for the operation of tracheotomy, as Boehme (p. 10) seems to believe. On the contrary. In spite of numerous ill successes, I hold to the principle, that where there is danger from suffocation through stenosis of the larynx, there is the indication for tracheotomy. Where there is no stenosis, I am glad not to operate. The results are not so bad, after all, when we remember that only such cases are operated upon which would be sure to die, if the operation were not performed. Even the number of children under two years saved by tracheotomy is increasing yearly. Krönlein reports 567 cases of diphtheria observed in the clinic of Berlin. Tracheotomy was performed in 504, with an average mortality of 70.8 per cent; the rate decreasing from 83.7 in 1870 to 61.81 in 1876. In the first year of life, the rate of deaths after tracheotomy was 93.3; in the second, 85.7; in the third, 80; and so on to 67.3; 66.6; 56; 76.4; 52.1; 53.5; 42.8; 66.6; 60 (in the twelfth year). The youngest child was 7 months old when it recovered. Of 85 under two years, 11 recovered.

The 400 cases of tracheotomy, reported by O. Wanscher as having been performed in Copenhagen from 1863 to 1876, yield even more favorable results. The rate of recoveries is as high as 42.1 per cent. He, too, finds the mortality increasing with the procrastination of the operation. Even infants under two years of age recovered, provided the operation was performed at an early period.

Of 50 cases of tracheotomy of Buchanan's (*Brit. Med. Jour.*, April 10th, 1880), 17 were classed as croup, and 33 as diphtheria, the latter including all those forms in which there was a distinct deposit of false white membrane on the tonsils, palate, or fauces. Of those 17 patients, 10 died, 1 of whom immediately after the operation, the

others in from 3 hours to 4 days. Of the 33 there was a mortality of 21, 1 of whom also died immediately after the operation, the others in from 6 hours to 13 days.

The indications, after the performance of the operation, in regard to the general process remain the same. Therefore, the general medicinal and dietetic treatment must be continued. Disinfection of the wound by zinc chloride, before the stitches are applied, is advisable. When the wound shows a diphtheritic appearance after twenty-four hours, sooner or later, or when the neighboring tissues swell, or when erysipelas shows itself, the stitches ought to be removed, and the wound treated with acid. carbolic. pur. and glycerine aa . While the disinfecting local treatment of the nose and pharynx is continued, a similar treatment is resorted to in regard to the trachea. I have atomized through the tube, in many cases, a two-per-cent solution of carbolic acid, every hour, every half-hour; in most cases, however, used a solution of carbolic acid in water ($\frac{1}{2}$ –5 per cent), or in lime-water, or lime-water with glycerine. This procedure has been kept up every quarter of an hour, every half-hour, every hour, for days in succession. The children are not annoyed by the proceeding and rarely get awake except from a severe attack of coughing. The atomizing is repeated very frequently, but a single compression of the balloon suffices for the purpose. For more direct and thorough applications—the tube either being removed for the purpose, or mostly through it—I use a long pigeon's or hen's feather, carefully examined before using, dipped in a solution of carbolic acid (1) to aq. calc. and glycerine (aa 5), or usually glycerine only (8–10). The amount of that liquid introduced into the trachea on that best of all instruments for the purpose: flexible, uninjurious, and effective, is sufficiently large, but not too large. A number of cases I have also exposed to a constant carbolic acid spray (2–3 per cent); a few I have treated with permanent turpentine inhalations as described above. All of them

were kept under the influence of steam. Thus surely Dr. Pauly is not correct when he attributes my acknowledged want of success with tracheotomy, during the last ten years, to my neglect of inhalations after the operation, nor was his prophesy, that the results would be better in future. I must admit that they have continued to be but unfavorable, though, having tracheotomized these twenty years more frequently, perhaps, than any physician in the States, I have been as anxious as any one to use every means in my power to disinfect.*

When, after the operation, the relief is next to none, particularly when the case takes a very rapid course, it is probably one of ascending croup which commenced in the trachea. Mechanical relief by pushing down the hen's feather, or a bundle of them, and turning it about and twisting must be tried. It is a much better instrument than pincers of all sorts and shapes. But what relief will be accomplished is of but very short duration. When fever will set in within a few hours, it means very much more frequently pneumonia than diphtheritic fever. It will soon be complicated by that disproportion between pulse and respiration so characteristic to inflammatory diseases. Then quinia in larger doses, 0.25, or 0.5 (grs. iv.-viii.) every two, four, eight hours, at the same time doses of sodium salicylate 0.25-0.40 (grs. iv.-vi.) every hour or two hours until the temperature goes down, small doses of digitalis, where the heart requires it, must be given at once. Procrastination is dangerous, the patients want careful watching, most of them die within two days after the operation.

The results of any treatment in membranous croup are

* Dr. Al. Hadden assures me that liq. subsulphat. ferri applied to the trachea, after the operation, has saved some of his cases. With boric acid, muriatic acid, bromid. ammon., aq. chlorin., pot. hypermangan., sod. salicyl., sod. sulpho-carbol., zinc. sulph.-carbol, I have not, like Krönlein, experimented. Like him, I have used alum, borax, pepsin, lime-water, but have in most cases proceeded as above described.

of so doubtful a character that any observation faithfully both made and reported may be of service. Bela Weiss relieved a boy of six years with undoubted croup by "massage," in the same manner in which he had previously removed the urgent symptoms of pseudo-croup. The child sat on the lap of the mother, who held his head backwards, the doctor, sitting in front of the patient, placed a hand on either side of the neck, interlocking four fingers of either hand—well oiled—posteriorly, prayer fashion, and moved his thumbs slowly and gently in the beginning, more forcibly afterward, from the horizontal ramus of the lower jaw down to the clavicle, raised the thumbs, and repeated the same manœuvre through five minutes and more. This operation was repeated a number of times in intervals of several hours, with favorable results. Respiration became easier, cough looser, membranes were expectorated. The author adds that cold applications, warm inhalations, a short time also potassium chlorate were resorted to in the usual manner, and recommends his treatment, not as a panacea, but as successful in that case and worthy of further trials.*

J. Szeparowicz (Centralb. f. Chir., No. 26, 1880) treated, in accordance with Schroetter's advice, a contraction of the larynx with bougies. A girl of four years had been tracheotomized for croup, eight months previously. The dilatation was persisted in for four weeks, with complete success.

Whether the symptoms arise from a marked degree of œdematous infiltration of the tissues, or from fibrinous exudation or degeneration of epithelium, need not concern us as far as tracheotomy comes into question. In the first class of cases, the after-treatment is more successful, because the trachea is more apt to be spared. Just as little need we consider whether, in individual cases, "the differ-

* *Casuistische Mittheilungen über die Anwendung der Massage bei Laryngitis catarrhalis und crouposa.* Arch. f. Kinderheilkunde, 1880, p. 201.

ential histological diagnosis has been made between diphtheria and croup."

DIPHTHERITIC PARALYSES.

The treatment of diphtheritic paralysis is simple enough in many cases; for sometimes nothing but patience and waiting are necessary. The limbs are usually restored to their normal condition, if the circumstances be in any way favorable. Anæmia and debility are invariable concomitants, and the diet and medical treatment must be regulated accordingly. We must not forget, however, that overfeeding and a sameness of diet are not permitted, for not rarely the muscular coat of the stomach suffers with the rest of the muscular tissue, and the secretion of gastric juice is very deficient in anæmic individuals. While therefore, from a therapeutic stand-point, iron is indicated, we must not neglect to pay particular attention to nutrition and digestion, and to aid the latter with pepsin and moderate amounts of muriatic acid, well diluted. Quinine and stimulants are appropriate wherever there is no contra-indication to their employment. The treatment of the paralysis itself, where it is not deemed judicious to wait, will naturally depend on the diagnosis of the condition in question. This alone can explain why various modes of treatment, the electric current among others, after being recommended by some authors, are branded by others. Where we have to deal with those rare changes in the brain and spinal cord, with apoplexy, "the utmost care is necessary" in order "not to make the condition still worse," and in such cases there would be a contra-indication to the use of the faradic current, but this would not hold true with regard to the use of the galvanic current in short sittings. Besides, central paralyzes are by no means so frequent as peripherous ones. In most cases, there is not the slightest elevation of temperature during the course of the paralytic phenomena. I lay great stress upon this point, for I am aware that many cases of central con-

gestions and inflammatory processes at times exhibit but very insignificant elevations of temperature. But as the diagnosis will depend on a positive knowledge of whether there have been changes of temperature, I rely on the rectal temperature only, for many a myelitis runs its course with no greater elevation above the normal than one-half or one degree. In all cases in which the temperature is normal or subnormal, I do not hesitate for a moment to employ the faradic or the galvanic current, according to circumstances. In addition to the internal administration of iron, I advise by all means the employment of *nux vomica*, in the form of strychnia. I cannot indorse Oertel's warning against the use of strychnia, on the ground that, as it acts centrally, it will positively give rise to an increased irritation of the morbid process in the spinal cord. The observations of a great many authorities, and my own which are rather extensive, cause me to look upon strychnia as the most reliable remedy in diphtheritic paralysis. Where there is no necessity for haste, we may give moderate doses, gradually increasing, in combination with iron; where there is danger in delay, it is more judicious to have recourse to subcutaneous injections, administered at regular intervals. Henoch has seen diphtheritic paralyses disappear in three weeks, under the use of hypodermic injections of strychnia. This, which has also been my experience on many occasions, corresponds with what Demme says (tenth report, 1873) in connection with the treatment of infantile paralysis. His statements I have seen verified in the latter disease, in cerebral paralyses and in diphtheritic paralysis. It also agrees with the favorable results from subcutaneous injections of strychnia in the temples in amaurosis, which Nagel was the first to witness, and which since have been observed by others, and by myself in several cases. I especially advocate the use of injections where there are urgent and dangerous paralytic manifestations, as in case of danger depending on the

paralysis of the muscles of deglutition and of respiration. Of course, where the former are affected, it is necessary to nourish the patient artificially, partly perhaps by nutrient enemata, but principally by means of the stomach-tube. In using the latter, it is unnecessary to introduce it into the stomach, as it only requires to be passed a few inches below the affected parts, when the œsophagus, far from manifesting the repugnance displayed by the pharynx, undertakes the further disposal of the food. In these cases, strychnia should be injected subcutaneously in the neck, once or twice daily. In a similar manner, it should be injected in the region of the chest, diaphragm, or neck, in paralysis of the respiratory muscles or of the glottis. In paralysis of the muscles of accommodation (in which Scheby-Buch claims to have seen the process cut short by the use of calabar bean, considered as inert by Hassner) they may be given in the forehead or temples.

Frictions dry and alcoholic, hot bathing, friction with hot water, kneading of the affected parts, will be found beneficial and pleasant.

DIPHTHERITIC CONJUNCTIVITIS

requires great attention and permits of no loss of time. Ice applications to the affected eye must be made constantly. Pieces of linen or lint kept on ice (better than in ice-water) of little more than the size of the eye, must be changed every minute or two, day and night. The danger to the cornea is so imminent that constant watchfulness is required. Boric acid in concentrated solution is, besides, dropped into the eye once every hour. The late Dr. H. Althof recommended and practised, where the rigidity of the eyelid was so great as to threaten rapid destruction of the cornea, a deep incision through the external angle to a distance of from one-half of an inch to an inch. Care must be taken that the well eye cannot get infected; for that purpose it is best to cover it with lint and collodion, or lint, or cotton, and adhesive plaster. Local infections

of the kind are very frequent. But lately have I observed a local infection even of the tongue from a diphtheritic eye.

CUTANEOUS DIPHTHERIA

requires the destruction of the membrane or the infected surface by carbolic acid—either concentrated or somewhat diluted with glycerine—or the application of the actual cautery. After that, the use of ice, or iced cloths, or diluted carbolic acid are indicated. As soon as the surface is no longer diphtheritic, the local and general treatment is to be continued on general principles.

SUMMARY.

Every case should be treated on general principles, with symptomatics, roborants, stimulants, febrifuges, externally, internally, or hypodermically.

The uncertainty of the termination, and the frequency of collapse or sepsis, prohibit procrastination. Waiting long means often waiting too long.

Alcohol (p. 157) is a very important adjuvant and remedy. The dose must often be apparently large, from two to twelve ounces daily, according to circumstances. Depletion is absolutely contra-indicated. Debilitating complications, such as diarrhea, must be stopped instantly.

Mouth and neck must be kept in a healthy condition. Stomatitis, chronic pharyngitis, hypertrophy of the tonsils, glandular enlargements must be relieved or removed preventively. Acute catarrh of mouth and pharynx requires the use of potassium or sodium chlorate (p. 159), in doses not exceeding a scruple daily for a child of a year, $1\frac{1}{2}$ –2 drachms for an adult. The single doses must be small and very frequent, every hour, half, or quarter hour. Large doses are dangerous, result often in nephritis, and have proved fatal.

The main indication in local diphtheria is local disinfection (p. 169). To disinfect the blood effectively we have no means. Salicylic acid changes into a salicylate which

is no longer a disinfectant. The amount of disinfectants required to destroy bacteria is so great that the living body could not endure them; for instance, carbolic acid, quinine, and sulphur. But the discipline of the house, school, and social intercourse can be so modified as to prevent the spreading of an epidemic. The instructions for disinfectants published by the National Board of Health (p. 175) are as simple as they are effective.

The inhalation of steam (p. 178) is very useful in catarrh of the respiratory organs, and also in inflammatory and diphtheritic affections. In fibrinous tracheo-bronchitis it has proved quite successful. But it may prove dangerous by excluding oxygen and overheating the room or tent. Drinking of large quantities of water (p. 181), with or without stimulants, also incites the action of the muciparous glands and aids in macerating membranes. The internal use of ice, and its local application to the affected parts, can be very useful. But the cases must be selected for each and any of the remedial agents and applications. The use of baths, and the cold or hot pack is controlled by general indications. The usefulness of lime-water (p. 183) and lactic acid has been greatly overestimated. Glycerine is a valuable adjuvant both externally and internally, but not more. Turpentine inhalations (p. 185) are deserving of further trials, though naturally they are more effective in purely inflammatory than in diphtheritic processes. Inhalations of ammonium chloride (p. 187) act favorably in catarrhal and inflammatory conditions, and deserve a trial for the purpose of aiding maceration of membranes. Mercurials (p. 188) are contra-indicated in the septic and gangrenous forms of diphtheria, but in those which assume more the purely inflammatory character with less constitutional debility and collapse, as in "sporadic croup," or in fibrinous tracheo-bronchitis, some reliable clinicians claim good results.

Astringents, such as tannin and alum, do not work favorably (p. 190).

Chloride of iron (p. 191) is amongst the most reliable antiseptic and astringent agents. Small doses in long intervals are quite useless. Moderate doses frequently repeated have a satisfactory general and local effect. A child of a year must take at least four grammes (a drachm) daily; a child of three or four years, from eight to fifteen grammes. The same or a larger dose for an adult. The chloride is to be mixed with water and glycerine in various proportions, so that a dose is taken every hour, every half-hour, every ten minutes. Thus the local applications to the throat become mostly superfluous. Potassium or sodium chlorate from two to four grammes (3 ss. -i.) daily may be added to advantage.

Carbolic acid is useful both in local and internal administration. According to the end to be reached, it may be used either in concentrated form, or in a one-per-cent solution (p. 196). Internally, in doses of a few grains to half a drachm daily.

Salicylic acid acts as a caustic when concentrated; in moderate solutions it destroys fetor; salicylates are antifebriles only (p. 197). The antifebrile effects of quinine are not so favorable in infectious as in inflammatory fevers; its antiseptic action is not satisfactory in practice (p. 198).

Deliquescent caustics are dangerous. Injury of the healthy mucous membrane must be avoided. Mineral acids, and particularly carbolic acid, when their application can be limited to the desired locality, are preferable (p. 200).

Bromine both internally and externally is warmly recommended by Wm. H. Thompson (p. 201).

Boric acid, in concentrated and milder solutions, has been recommended as a local application to membranous deposits generally, and to the diphtheritic conjunctiva in particular (p. 206).

Sodium benzoate does not deserve the eulogies bestowed on it from theoretical reasoning (p. 207).

Eucalyptus, sulphur, copaiba, and cubeb cannot be recommended (p. 209).

Membranes must not be torn off, and not removed unless they are nearly detached. Caustics are contra-indicated except where their application can be limited to the diseased surface. No healthy part must be injured. Swelled lymph-glands require ice, iodine, iodoform, mercury, poultices, incision, carbolic acid, according to circumstances (p. 216), and at all events frequent and careful disinfection of the mucous membrane from which their irritation originates. Diphtheria of the nose (p. 217) is apt to be fatal unless careful treatment is commenced at once. It consists of persistent disinfection of the nares and pharynx by means of injections. The tendency to sepsis forbids a long intermission of them. They must be continued day and night for one or several days, no matter whether the glandular swelling is considerable or not.

Laryngeal diphtheria (p. 222) proves fatal in almost every case, unless tracheotomy be performed. It is the less successful the more the epidemic or case bears a septic character. Emetics, such as zinc, copper, or turpeth mineral, are useful for the removal of half detached membranes.

Diphtheritic paralysis (p. 228) requires good and careful feeding, iron, strychnia, the faradic or galvanic currents, friction, hot bathing. Urgent cases indicate the hypodermic administration of strychnia.

Diphtheritic conjunctivitis is benefited (p. 230) by ice and boric acid; cutaneous diphtheria, by local cauterization and disinfection, besides general treatment.

INDEX.

- Abscesses of lymphatic glands in diphtheria, 116.
Absence of acinous glands to a certain extent prevents diphtheria of the vocal cords, 128.
Adynamia in diphtheria, 51.
Aëtius, 1.
Age when diphtheria occurs, 50.
Air passages, anatomical appearances in the, table showing the, 126.
Albuminous urine produced by chlorate of potassium, 166.
Albuminuria in diphtheria, 51, 55, 90, 106, 136, 144.
 of scarlatina and diphtheria, diagnosis between, 90.
Alum in the treatment of diphtheria, 190.
America, first known case of diphtheria in, 3.
Ammonium chloride in the treatment of diphtheria, 187.
Anatomical appearances in diphtheria, 108 *et seq.*
 appearances of the epiglottis, 124.
 appearances of the mucous membrane of the mouth, 122.
 appearances of the mucous membrane of the nasal cavities, 123.
 appearances of the mucous membrane of the trachea and bronchi, 125.
 appearances, table showing the, in the air passages, 126.
Anæmia from diphtheritic paralysis, 228.
Angina ulcusculosa, 4.
Animals causing diphtheria in human beings, 65.
 with diphtheritic affections, 65.
Antimonials in diphtheritic laryngitis, 223.
Antiseptic nature of chloride of iron, 194.
Antiseptics in diphtheria, 220.
Aphonia in laryngeal diphtheria, 137.
Applications, local, beneficial in nasal diphtheria, 220, 221.
Aqua calcis in diphtheria, 211.
Aretæus, 1.
Artificial diphtheritic membrane, 111.
Arytenoid cartilages, cicatrization of, a sequel of diphtheria, 73.
Asclepiades, 1, 2.
Aspergillus nigrescens, 66.
Asthenic and suffocative forms of diphtheria, 2.
Astringents in the treatment of diphtheria, 190.

- Ataxia, locomotor, in diphtheria, 101.
Atomizer in treatment of diphtheria, 179.
 use of, in nasal diphtheria, 219.
Autopsies in cases of diphtheritic paralyses, 103.
Autopsy, first, in diphtheria, 3.
Author's views as to origin of diphtheria, 54, 55.
- Bacteria, 170.
 destruction of, to prevent diphtheria, 174.
 experiments as to the methods of destroying, 174.
 in diphtheria, 49, 50, 111.
 not always found in diphtheria, 66.
 putrefaction without, 39.
 relation of, to diphtheria of the intestine, 117.
- Balano-posthitis, diphtheritic, 89.
Ballonius, 2.
Balsamica, the, in diphtheria, 209.
Balsamics, the, nephritis following the use of, 210.
Bard, 10, 165.
Bastian, 23.
Baths in diphtheria, 154.
Benzoate of sodium in diphtheria, 170, 206.
Bite causing diphtheria, 2.
Bladder, diphtheria of the, 87.
Bleeding in diphtheria, 157.
Blood, the, in diphtheria, 92.
Boric acid and iodine injections in nasal diphtheria, 220.
 acid in diphtheria, 206.
 acid in diphtheritic conjunctivitis, 230.
Bougies used to remove contraction of larynx, 227.
Brandy in diphtheria, 157.
Bretonneau, 12, 13, 14, 16, 18, 165.
Bromine in diphtheria, 200.
Bronchi and trachea, mucous membrane of the, anatomical appearances of the, 125.
Bronchitis in children, 27.
Broncho-pneumonia in diphtheria, 80.
- Cælius Aurelianus, 1.
Calomel in diphtheria, 157, 189, 211.
Calves affected by diphtheritic poison, 65.
Carbolated sulphate of sodium in diphtheria, 211.
Carbolic acid, amount required to disinfect, 174.
 acid as a disinfectant, 175, 195.
 acid in the destruction of bacteria, 174.
 acid in diphtheria, 195, 214.
 acid spray after tracheotomy, 225.

- Catarrhal laryngitis, 138.
- Catarrh, chronic, after diphtheria, 104.
 chronic nasal, and so-called scrofula, 131.
 nasal, complicated with diphtheria, 72.
 nasal, followed by nasal diphtheria, 217.
- Catheterization in diphtheritic laryngitis, 223.
- Causes of diphtheria, 2, 25, 51, 52, 53, 61, 85.
 of frequency of diphtheria in children, 30.
 of pneumonia in diphtheria, 81.
 predisposing, of diphtheria, 32.
- Caustic action of salicylic acid, 197.
- Cauterization in diphtheria, 211.
 of diphtheritic membranes to be thoroughly done to be effective, 200.
- Central origin of paralysis, 97.
- Chemical nature of diphtheritic poison, 50.
- Childhood, diphtheria a disease of, 49.
- Children, bronchitis in, 27.
 causes of frequency of diphtheria in, 30.
 infectious diseases in, 29.
- Chloral in diphtheria, 213.
- Chlorate of potassium, action of, upon the heart, 162.
 of potassium causing cyanosis, 163, 165.
 of potassium causing death, 163 *et seq.*
 of potassium causing nephritis, 163, 165.
 of potassium, convulsions produced by, 163.
 of potassium, danger of administration in large doses, 162.
 of potassium in the treatment of diphtheria, 160.
 of potassium producing albuminous urine, 166.
 of potassium and of sodium, prophylactics in diphtheria, 159.
- Chloride of ammonium in the treatment of diphtheria, 187.
 of iron, experiments with, 193.
 of iron in the treatment of diphtheria, 191, 211.
- Chlorine in diphtheria, 213.
- Chromic acid in diphtheria, 199.
- Cicatrizization of arytenoid cartilages a sequel of diphtheria, 73.
- Ciliated epithelium less liable to be affected in diphtheria, 127.
- Circumcision-wounds, diphtheritic infection of, 88.
- Clothing of diphtheritic patients should be disinfected, 177.
- Cocco-bacteria dissolved and eliminated, 41.
 in the living blood not proven, 41.
- Colden, Cadwallader, 7.
- Collapse and death in mild cases of diphtheria, 132.
- Communicability of diphtheria, 52.
- Complications, danger of, in diphtheria, 152.
- Congestion of the pharynx may be either traumatic or diphtheritic, 134.

- Conjunctivitis, diphtheritic, 17, 73, 143.
 diphtheritic, treatment of, 230.
 Constitutional nature of diphtheria, 52, 56.
 Contagiousness of diphtheria, 51, 57, 67.
 Contagium animatum, 36.
 Contraction of larynx relieved by bougies, 227.
 Convulsions a bad symptom in diphtheria, 70.
 produced by chlorate of potassium, 163.
 Copaiba in diphtheria, 209.
 Copperas solution to disinfect discharges from diphtheritic patients, 176.
 Corpses, manner of preparing, of diphtheritic patients after death, 177.
 Croup and diphtheria, symptoms compared, 139 *et seq.*
 and laryngeal diphtheria, identity or non-identity of, 112.
 ascending, 142.
 "massage" for relief of, 227.
 membranous, and diphtheritic laryngitis, 138.
 membranous, treatment of, 222.
 occurring when diphtheria is prevalent, 77, 78.
 prognosis in, 138.
 Croupous deposits in diphtheria, 70.
 diphtheria, 104.
 laryngitis and laryngeal diphtheria, 119.
 membrane formed by migration of white blood-corpuscles, 110.
 Cubebs in diphtheria, 209.
 Curtis and Satterthwaite, 23, 43.
 Cutaneous diphtheria, treatment of, 231.
 Cyanosis an unfavorable sign in diphtheria, 153.
 resulting from an over-dose of chlorate of potassium, 163, 165.
 ushers in death in tracheal diphtheria, 143.

 Danforth, 3.
 Danger of allowing children affected with nasal diphtheria to sleep, 220.
 of complications in diphtheria, 152.
 of conveying diphtheritic poison during an epidemic, 158.
 of incisions for rigidity of the eyelid in diphtheritic conjunctivitis, 230.
 Dead bodies of diphtheritic patients to be kept moist, 173.
 Death caused by chlorate of potassium, 163 *et seq.*
 -rate in diphtheria, 157.
 sudden, in mild cases of diphtheria, 132.
 Debility from diphtheritic paralysis, 228.
 Deformity of penis caused by diphtheria, 90.
 Deglutition, difficulty of, caused by paralysis, 99.
 Deodorizers, definition of, 175.
 Deposits in diphtheria, 69.
 Destruction of the eye in diphtheria, 73, 105.
 Diagnosis between albuminuria of scarlatina and diphtheria, 90.

- Diagnosis between enteritis and diphtheria, 84.
 - between stomatitis and diphtheria, 134.
 - of diphtheria, 134.
 - of membranous laryngitis, 137.
 - of myelitis in diphtheria, 229.
- Diagnostic feature in diphtheritic tracheo-bronchitis, 79.
- Diarrhoea, treatment of, in diphtheria, 158.
- Digestion interfered with by chlorate of potassium, 162.
- Digitalis after performance of tracheotomy, 226.
- Diphtheria a constitutional disease, 56.
 - a disease of childhood, 49.
 - after circumcision, 88.
 - after urethrotomy and lithotomy, 88.
 - age when it occurs, 50.
 - albumen in urine in, 51, 55, 90, 136, 144.
 - an adynamic disease, 51.
 - anatomical appearances in, 108 *et seq.*
 - and croup, symptoms compared, 139 *et seq.*
 - and erysipelas, relations between, 86.
 - and filth, 34.
 - and milk, 63.
 - and scarlatinal albuminuria, diagnosis between, 90.
 - and stomatitis, diagnosis between, 134.
 - antiseptics in, 220.
 - apt to recur, 50.
 - at different ages, 30.
 - author's views as to origin of, 54, 55.
 - bacteria in, 49, 50.
 - broncho-pneumonia in, 80.
 - causes of, 2, 25, 51, 52, 53, 61, 85.
 - causes of frequency of, in children, 30.
 - causes of pneumonia in, 81.
 - causing deformity of penis, 90.
 - changes of temperature in, 33.
 - cicatrizization of arytenoid cartilages a sequel of, 73.
 - ciliated epithelium less liable to be affected in, 127.
 - communicability of, 52.
 - communicated by animals, 64, 65.
 - communicated by kissing, 58.
 - complicating various diseases, 72, 81, 82.
 - considered by some a parasitic disease, 23.
 - considered by some a self-limited disease, 156.
 - constitutional symptoms in, 51.
 - contagiousness of, 51, 67.
 - convulsions and vomiting bad symptoms in, 70.
 - croupous deposits in, 70.

- Diphtheria, danger of complications in, 152.
diagnosis between enteritis and, 84.
diagnosis of, 134.
diagnosis of myelitis in, 229.
difference in invasion of, 54.
disinfectants in, 220.
division of remedies in, 178.
duration of incubation of, 51, 59, 60.
duration of prodromal stage of, 68.
ecchymoses in, 91.
emboli in, 115.
endemic causes of, 62.
epidemic nature of, 51, 61.
epistaxis in, 145.
etiology of, 27.
excrements to be removed in, 173.
fever not always a prominent symptom in, 135.
fibrinous pneumonia in, 80.
first autopsy in, 3.
first known case in America, 3.
first seen in respiratory passages, 53.
forms of, 104.
frequency of, 49.
frequency of relapses in, 32.
gangrene in, 71, 145.
has a predilection for certain organs and localities, 120, 133.
heart affections in, 91.
history of, 1.
identity of all forms of, 10.
immunity from, 50.
incubation of, in animals, 65.
infectious nature of, 58.
inflammatory affections of the lungs in, 80.
influence of season on, 33.
infrequent from third to seventh month, 31.
in France, 10.
in Holland, 10.
in New England, 3, 4.
in Sweden, 10.
in Switzerland, 10.
insidious nature of, 53.
intestinal, 84.
in the fowl, 66.
isolation in, 158, 171.
laryngeal, and croup, identity or non-identity of, 112.
laryngeal, and croupous laryngitis, 119.

- Diphtheria, laryngeal and tracheal, local in character, 128.
 laryngeal, in the adult, 27.
 leucocytes in, 113, 114.
 locomotor ataxia in, 101.
 lung affections in, 115.
 manifestations of, 69, 70.
 manner of infection in, 51.
 mechanical removal of membranes in, 214.
 micrococci as carriers of, 24.
 mild cases of, sudden collapse and death in, 132.
 mitral valve affected in, 91.
 mortality in, 150.
 nasal, 72.
 nasal, sleep dangerous in, 220.
 nasal, very fatal, 129, 133.
 not necessarily caused by polluted water, 62.
 nourishment, 162.
 often accompanied by pharyngitis, 53.
 of the vocal cords, danger of suffocation in, 129.
 of the vocal cords, general infection uncommon in, 128, 129.
 or muguet, 134.
 paralysis in, 52, 73, 96, 97.
 parasitic nature of, not proven, 49.
 pavement epithelium liable to be affected in, 127.
 pharyngeal membrane often first affected in, 53.
 predisposing causes of, 32.
 primary, of the trachea, 142.
 prognosis in, 149 *et seq.*
 prone to affect mucous membranes, 121.
 prophylaxis and prophylactics in, 58, 158, 159, 172.
 pseudo-leukæmia in, 92.
 sequelæ of, 73.
 severity of, dependent upon the vascular and lymphatic systems, 129.
 severity of paralysis in, 97.
 sewers and, 34.
 sex in, 29.
 skin eruption in, 144.
 sometimes limited to certain localities, 121.
 strabismus in, 10.
 suffocative and asthenic forms of, 2.
 swelling of the glands in, 131, 133.
 symptoms of, 68.
 synonyms of, 1.
 temperature in, 55, 68.
 the blood in, 92.
 the soft palate and pharynx in, 69.

- Diphtheria, thrombi in, 91.
 tonsils in, 30.
 treatment of, 154 *et seq.*
- Diphtheritic affections in animals, 65.
 fever, 91.
 laryngitis and membranous croup, 138.
 membrane, 70.
 membrane, artificial, 111.
 membrane, character of, determined by amount of elastic tissue,
 number of muciparous glands, etc., 122.
 membrane does not originate in epithelium according to some, 112.
 membranes, cauterization of, to be thoroughly done to be effective,
 200.
 membranes on the vocal cords not easily thrown off, 129.
 membranes to be rendered innocuous, 199.
 membranes, various methods of dissolving, 183, 184, 188, 189.
 patients, their corpses should be kept moist, 173.
 paralyses, 2, 96, 106, 145.
 paralyses, causes of, 98.
 paralyses do not run a certain course, 97.
 paralyses, electrical applications in, 103.
 paralyses, statistics of, 99.
 poison, chemical nature of, 50.
 poison, danger of conveyance of, during an epidemic, 158.
 poison impeded by the mucous glands, 127.
 poison, manner of entrance into system, 52.
 process affected by the character of the surface, 124.
 process, doctrines as to the, 109 *et seq.*
 process, statistics of the, 114.
 tracheo-bronchitis, 77.
- Discharges from diphtheritic patients to be received in vessels containing copperas
 solution, 176.
- Disinfectant properties of carbolic acid, 174, 195.
 properties of salicylic acid, 197.
- Disinfectants eighty years ago, 9.
 in diphtheria, 220.
 mode of using, 176.
- Disinfection of the clothing of diphtheritic patients, 177.
 of the wound caused by tracheotomy, 225.
 to be enforced by authorities in an epidemic of diphtheria, 172.
 to prevent diphtheria, 173.
- Douglass, 3.
- Duration of incubation of diphtheria, 59, 60.
- Drainage, bad, a cause of diphtheria, 61.
- Dyspnœa in laryngeal diphtheria without accompanying formation of membrane,
 137.

- Ecchymoses in diphtheria, 91.
- Elastic tissue, amount of, determines the character of the diphtheritic membrane in some cases, 122.
- Electrical applications in diphtheritic paralysis, 103.
- Emetics in diphtheritic laryngitis, 223.
- Emboli in diphtheria, 115.
- Endemic causes of diphtheria, 62.
- Endocarditis, diphtheritic, 115.
- Endogenous formation of pus globules in epithelium doubted, 110.
- Enteric fever complicated with diphtheria, 81, 82.
- Enteritis, diagnosis between diphtheria and, 84.
- Entrance of infectious material into the system, mode of, 130.
- Epidemic influences causing diphtheria, 53.
 - nature of diphtheria, 51, 61.
- Epiglottis, anatomical appearances of the, 124.
 - diphtheria of the, 76.
- Epistaxis in diphtheria, 145.
- Epithelial cells, changes in, considered by some characteristic of diphtheria, 109.
 - changes in membrane in diphtheria, 108.
- Epithelium, ciliated, less liable to be affected in diphtheria, 127.
 - endogenous formation of pus globules in, doubted, 110.
 - pavement, most liable to be affected in diphtheria, 127.
 - the, according to some, is not the source of origin of diphtheritic membrane, 112.
- Eruptions of the scalp, treatment of, in diphtheria, 158.
 - on the skin in diphtheria, 144.
- Erysipelas and diphtheria, relations between, 86.
- Etiology of diphtheria, 27.
- Eustachian tubes, diphtheria of, 74.
- Excrements to be removed in diphtheria, 173.
- Exhaustion in diphtheria, 155.
- Experiments with chloride of iron, 193.
- Exposure during tracheotomy causing diphtheria, 59.
- Extremities, upper and lower, paralysis of the, 100.
- Eye, destruction of the, in diphtheria, 105.
 - diphtheria of the, 73.
- Eyelids, rigidity of, in diphtheritic conjunctivitis, 230.
- Fever, diphtheritic, 91.
 - enteric, complicated with diphtheria, 81, 82.
 - high, obscures diagnosis, 135.
 - not always a prominent symptom in diphtheria, 135.
 - relative absence of, pathognomonic of diphtheritic laryngitis, 138.
- Fibrinous pneumonia in diphtheria, 80.
- Filth and diphtheria, 34, 50.
- First known case of diphtheria in America, 3.

- Fowl affected with diphtheria, 66.
Frequency of diphtheria, 10, 49.
 of diphtheria and pharyngitis, 53.
 of diphtheria in children, causes of, 30.
 of diphtheritic conjunctivitis, 74.
 of relapses in diphtheria, 32.
Frictions in diphtheritic paralyses, 230.
Fumigation by means of roll sulphur, manner of, 176.

Galen, 1.
Gangrene in diphtheria, 71 145.
"Garget," differential diagnosis of, 64.
Gargles in diphtheria, 212.
 of chloride of iron not useful in diphtheria, 192.
Gas prevents diphtheria, 58.
Geddings, 16.
Genito-urinary organs, diphtheria of the, 86.
Glands, absence of acinous, prevents diphtheria of the vocal cords in many cases,
 128.
 lymphatic, appearances of, in diphtheria, 116.
 mucous, impede the action of diphtheritic poison, 127.
 swelling of the, in diphtheria, 131, 133.
 treatment of swelling of lymphatic, 216.
Glandular swelling, diminution of, a good sign, 220.
 swellings, not always marked in diphtheria, 104.
Glycerin as a solvent of diphtheritic membranes, 183.
Graefe, 17.
Granular infiltration characteristic of diphtheria, 108.
Granulation tissue, changes in, in diphtheria, 108.

Hallier, 23, 47.
Heart, action of chlorate of potassium upon, 162.
 affections in diphtheria, 91, 114.
Henle, 36.
History of diphtheria, 1.
Hodgkin's disease occurring in diphtheria, 92.
Holland, diphtheria in, 10.
Home, 10.
Hüter, 109.
Human beings inoculated with diphtheria by animals, 65.
Hydrargyrum in the treatment of diphtheria, 183.
Hypodermic injections of mercury in diphtheria, 190.

Ice in diphtheria, 182, 230.
Identity of all forms of diphtheria, 10.
 or non-identity of laryngeal diphtheria and croup, 112.

- Immunity from diphtheria, 50.
- Incisions, danger of, for relief of rigidity of eyelid in diphtheritic conjunctivitis, 230.
- Incubation of diphtheria, duration of, 51, 59, 60, 67.
 - of diphtheria in animals, 65.
- Infection, general, a cause of diphtheria, 61.
 - general, in diphtheria of the vocal cords uncommon, 129.
 - of diphtheria, manner of, 51, 58.
- Infectious diseases in children, 29.
 - material, mode of entrance of into the system, 130.
 - nature of diphtheria, 58.
- Infiltration, granular, characteristic of diphtheria, 108.
- Inflammatory affections of the lungs in diphtheria, 80.
- Influenza complicated with diphtheria, 72.
- Inhalations in diphtheria, 212.
 - of steam in diphtheria, 178, 181.
 - of turpentine in the treatment of diphtheria, 185, 209.
- Injections in nasal diphtheria sometimes fatal, 218.
 - of strychnia in diphtheritic paralysis, 229.
- Inoculability of diphtheria, 51.
- Insidious nature of diphtheria, 53.
- Insufflation of sulphur in diphtheria, 209.
- Intestinal canal, appearances of, in diphtheria, 116.
- Intestine, diphtheria of the, 84, 105.
- Invasion of diphtheria, difference in, 54.
- Iodine and boracic acid injections in nasal diphtheria, 220.
 - ointments for swelling of the glands, 216.
- Ipecac in diphtheritic laryngitis, 223.
- Iron as a nervous stimulant, 195.
 - increases the power of oxydation, 195.
 - in the treatment of diphtheria, 176, 191, 193, 194, 211.
- Isambert, 18.
- Isolation in diphtheria, 158, 171.
- Josselyn, 3.
- Jurine, 12.
- Kidneys, appearances of, in diphtheria, 116.
 - diphtheria of the, 90.
 - micrococci in the, 46.
- Kiss, a, communicating diphtheria, 58.
- Klebs, 23, 48.
- Lactic acid as a solvent of diphtheritic membranes, 183, 184.
- Laryngeal diphtheria, 136.
 - diphtheria in the adult, 27.
 - diphtheria, local in character, 128.

- Laryngeal diphtheria more frequent in boys, 50.
- Laryngitis, catarrhal, 138.
 diphtheritic, and membranous croup, 138.
 diphtheritic, treatment of, 222.
 membranous, diagnosis of, 137.
- Laryngoscopic examination in diphtheria, 136.
- Larynx, contraction of, relieved by bougies, 227.
 membranous deposits in, 138.
- Le Cat, 9.
- Letzerich, 48, 82.
- Leukocytes in diphtheria, 113, 114.
- Leukocythæmia in diphtheria, 92.
- Lime-water in diphtheria, 183, 211.
- Lithotomy causing diphtheria, 88.
- Liver, appearances of, in diphtheria, 116.
- Local applications beneficial in nasal diphtheria, 220, 221.
 origin of diphtheria, 52, 53.
- Locomotor ataxia in diphtheria, 101.
- Lugol's solution in diphtheria, 211.
- Lung affections in diphtheria, 80, 115.
- Lungs and trachea, mycosis of the, 65.
- Lymphadenitis generally severe in nasal diphtheria, 136.
 gives rise to sudden elevation in temperature, 135.
 not necessary to diagnose a case of diphtheria, 136.
- Lymphatic and vascular systems, severity of diphtheria dependent upon the, 129
 glands, appearances of, in diphtheria, 116.
 glands, treatment of swelling of, 216.
- Lymphatics, effect of chloride of iron on the, 193.
- Maingault, 17.
- Manifestations of diphtheria, 69, 70.
- "Massage" for relief of croup, 226.
- Membrane, artificial diphtheritic, 111.
 diphtheritic, 70.
 diphtheritic, according to some authorities does not originate in
 thelium, 112.
 formation of, in diphtheria sometimes prevented by chlor
 potassium and sodium, 160.
 the, a characteristic sign in diphtheria, 108, 134.
- Membranes in intestinal diphtheria, 85.
 mechanical removal of, in diphtheria, 214.
 mucous, prone to be affected by diphtheria, 121.
- Membranous croup or diphtheritic laryngitis, 138.
 croup, treatment of, 222.
 deposits in larynx, 138.
 laryngitis, diagnosis of, 137.

- Mercado, 2.
- Mercury given hypodermically in diphtheria, 190.
 given to liquefy diphtheritic membranes, 188, 189.
 in the treatment of diphtheria, 187.
 not a specific in diphtheria, 188.
 oleate of, in diphtheria, 190.
 sulphate of, in diphtheritic laryngitis, 223.
 to be given in small doses frequently to produce effect in diphtheria, 189.
- Micrococci, 194.
 as carriers of diphtheria, 24.
 disappear as erysipelatous process progresses, 40.
 excite no reaction, 25.
 in the kidneys, 46.
- Micro-organisms and septic poisons, no etiological connection between, 38.
- Middleton, 11.
- Milk a cause of diphtheria, 63.
- Mitral valve affected in diphtheria, 91.
- Mortality in diphtheria, 150.
- Mouth, diphtheria of the, 81, 105.
 mucous membrane of, anatomical appearances of, in diphtheria, 122.
- Muciparous glands, determining the character of diphtheritic membrane in certain cases, 122.
- Muco-salivary diphtheritis caused by extirpation of the tongue, 85.
- Mucous glands impede the action of diphtheritic poison, 127.
 membrane of the mouth, anatomical appearances of, 122.
 membrane of the nasal passages, anatomical appearances of, 123.
 membrane of the trachea and bronchi, anatomical appearances of the, 125.
 membranes prone to be affected by diphtheria, 121.
- Muguet or diphtheria, 134.
- Mycosis of the trachea and lungs, 65.
- Myelitis, diagnosis of, in diphtheria, 229.
- Mykosis oesophagi, 82.
- Nasal catarrh complicated with diphtheria, 72.
 catarrh, treatment of, during epidemics of diphtheria, 158.
 cavities affected in diphtheria, 71.
 cavities, mucous membrane of, anatomical appearances of, 123.
 diphtheria, 72.
 diphtheria following nasal catarrh, 217.
 diphtheria, lymphadenitis generally severe in, 136.
 diphtheria, sleep dangerous in, 220.
 diphtheria, treatment of, 217.
 diphtheria, very fatal, 129, 133.
- Necrotic diphtheria, 104.
- Nephritis following the use of the balsamics, 210.

- Nephritis resulting from an overdose of chlorate of potassium, 163, 165.
 Nervous system, affections of, in diphtheria, 93, 106.
 tissue, anomalies of nutrition of the, causing diphtheritic paralysis, 98.
 Neurin as a solvent of diphtheritic membranes, 184.
 New England, diphtheria in, 3, 4.
 Nitrate of silver in the treatment of diphtheria, 190, 199.
 Nostrils of attendants on diphtheritic patients to be filled with cotton, 173.
 Nourishment in diphtheria, 154, 162.
 Nurses of diphtheritic patients to wear cotton in their nostrils, 173.
 Nutrition, anomalies of, of nervous tissue causing diphtheritic paralysis, 98.

 Œdema glottidis detected by palpation, 137.
 glottidis, general, uncommon in diphtheria, 137.
 glottidis in diphtheria, 76, 105.
 Oertel, 37, 38, 78, 155, 190, 229.
 Œsophagus, diphtheria of the, 82.
 Ogden, Jacob, 9.
 Oleum eucalypti e foliis in diphtheria, 209.
 Origin, local, of diphtheria, 52, 53.
 of diphtheria, author's views of the, 54, 55.
 of diphtheritic paralysis, 96.
 Otitis in diphtheria, 74.
 Oxydation increased by iron, 195.
 Ozone in diphtheria, 205.

 Palate, soft, in diphtheria, 69.
 Panum, 38, 46.
 Parasitic nature of diphtheria not proven, 23, 49.
 Paralyzes, diphtheritic, treatment of, 228.
 peripheral, more frequent than central, 228.
 Paralysis, diphtheritic, 2, 52, 96, 142.
 diphtheritic, statistics of, 99.
 of central origin, 97.
 of the lower and upper extremities, 100.
 of the pneumogastric nerve in diphtheria, 96.
 of the sense of taste in diphtheria, 97.
 of the vocal cords, 138.
 severity of, in diphtheria, 97.
 Paresis of the thyro-arytenoid muscles a sequel of diphtheria, 73.
 Pathological appearances caused by diphtheritic endocarditis, 115.
 Pavement epithelium altered in diphtheria, 108.
 epithelium most liable to be affected in diphtheria, 127.
 Penis, deformity of, caused by diphtheria, 90.
 Pepsin as a solvent of diphtheritic membranes, 184.
 Perforation and destruction of the eye in diphtheria, 74.
 Peripheral more frequent than central paralyzes, 228.

- Pharyngeal congestion may be either traumatic or diphtheritic, 134.
 membrane often first affected in diphtheria, 53.
- Pharyngitis and diphtheria, 53.
 occurring during diphtheria epidemics, 159.
- Pharynx in diphtheria, 68.
 often not affected in diphtheritic tracheo-bronchitis, 79.
- Placental diphtheria, 88.
- Phlegmon, incision of, resulting in diphtheria, 52.
- Pneumogastric nerve, paralysis of the, in diphtheria, 96.
- Pneumonia, causes of, in diphtheria, 81.
 diphtheritic, 143.
 fibrinous, in diphtheria, 80.
- Polluted water not necessarily a cause of diphtheria, 62.
- Potassa in diphtheria, 199.
- Potassium chlorate a prophylactic in diphtheria, 159.
 chlorate, effects of, in diphtheria, 160 *et seq.*
- Powers' investigation as to communicability of diphtheria by means of milk, 64.
- Predisposing causes of diphtheria, 32, 50, 58.
- Premises, the, should be disinfected with copperas solution, 177.
- Prevention of diphtheria, 158, 173.
- Primary diphtheria of the trachea, 142.
- Prodromal stage, duration of, of diphtheria, 68.
- Prognosis in croup, 138.
 in diphtheria, 149.
- Prophylactic agents in diphtheria, 58, 159.
- Pseudo-croup, 138.
- Pus globules, endogenous formation of, in epithelium doubted, 110.
- Putrefaction without bacteria, 39.
- Quinia after the performance of tracheotomy, 226.
 in diphtheria, 154, 171, 198, 213, 228.
- Recurrence of diphtheria, 50.
- Relapses frequent in diphtheria, 32.
- Relations between erysipelas and diphtheria, 86.
- Removal, mechanical, of membranes in diphtheria, 214.
- Remedies in diphtheria, division of, 178.
- Respiratory muscles, paralysis of the, most dangerous, 101.
 organs affected in diphtheria, 75.
 passages, diphtheria first seen in the, 53.
- Rhinoscopic examination in diphtheria, 136.
- Rigidity of the eyelid in diphtheritic conjunctivitis, 230.
- Rokitansky, 81.
- Salicylate of sodium in diphtheria, 197, 211.
- Salicylic acid as a caustic, 197.

- Salicylic acid, disinfectant properties of, 197.
 - acid in diphtheria, 170, 196.
- Satterthwaite and Curtis, 23, 43.
- Scarlatinal and diphtheritic albuminuria, diagnosis between, 90.
- Schools, necessity of closing them in diphtheria epidemics, 172.
- Sclerotic, destruction of, in diphtheria, 105.
- Scrofula, so-called and chronic nasal catarrh, 131.
- Season, influence of, on diphtheria, 33.
- Self-limitation of diphtheria, 156.
- Sensitive paralyses, 101.
- Sequelæ of diphtheria, 73.
- Severity of diphtheria dependent upon the vascular and lymphatic systems, 129.
 - of paralysis in diphtheria, 97.
- Sewers and diphtheria, 34.
- Sex in diphtheria, 29.
- Silver nitrate in diphtheria, 199.
- Skin, diphtheria of the, 2, 86.
 - diphtheritic poison introduced by the, 52.
 - eruption in diphtheria, 144.
- Sleep dangerous in nasal diphtheria, 220.
- Sodii sulphatis carbolatæ in diphtheria, 211.
- Sodium benzoate in diphtheria, 170, 206.
 - chlorate a prophylactic in diphtheria, 159, 160.
 - salicylate of, in diphtheria, 154, 197, 211.
- Source of diphtheria, 50.
- Spleen, appearances of, in diphtheria, 116.
- Statistics of diphtheritic paralysis, 99.
 - of the diphtheritic process, 114.
 - of tracheotomy in diphtheritic laryngitis, 224.
- Steam, inhalations of, in diphtheria, 178, 181.
- Stimulants in diphtheria, 156.
- Stomach, diphtheria of the, 82.
- Stomatitis and diphtheria, diagnosis between, 134.
 - follicularis not to be confounded with diphtheria, 134.
 - occurring during diphtheria epidemics, 159.
- Strabismus in diphtheria, 10.
- Strychnia in diphtheritic paralyses, 229.
- Suffocation, danger of, in diphtheria of vocal cords, 129.
- Suffocative and asthenic forms of diphtheria, 2.
- Sulphurous acid in the destruction of bacteria, 174.
- Sulphur, roll, for fumigation, 176.
 - insufflation of, in diphtheria, 209.
- Summary of anatomical appearances in diphtheria, 132.
 - of diagnosis of diphtheria, 147.
 - of etiology of diphtheria, 49.
 - of history of diphtheria, 25.

- Summary of manner of infection in diphtheria, 56.
 - of symptoms of diphtheria, 104.
 - of treatment of diphtheria, 231.
- Suppuration rare when glands swell in diphtheria, 217.
- Sweden, diphtheria in, 10.
- Swelling of the glands in diphtheria, 131, 133.
 - of the glands in diphtheria, treatment of, 216.
- Switzerland, diphtheria in, 10.
- Symptoms of croup and diphtheria compared, 139 *et seq.*
 - of diphtheria, 51, 68.
 - of laryngeal diphtheria, 77.
- Synonyms of diphtheria, 1.

- Table showing the anatomical appearances in the air passages in diphtheria, 126.
- Tannin in the treatment of diphtheria, 190, 211.
- Taste, sense of, paralysis of, in diphtheria, 97.
- Temperature in diphtheria, 33, 55, 68.
- Thompson, W. H., on the effects of bromine in diphtheria, 200 *et seq.*
- Throat distemper, 5, 7.
 - the, in diphtheria, 70.
- Thrombi in diphtheria, 91.
- Tissues acting differently after inoculation, 42.
- Tommasi, 109.
- Tongue, extirpation of the, a cause of muco-salivary diphtheritis, 85.
- Tonics in diphtheria, 155.
- Tonsils in diphtheria, 30, 215.
- Trachea and bronchi, mucous membrane of the, anatomical appearances of the, 125.
 - and lungs, mycosis of the, 65.
 - primary diphtheria of the, 142.
- Tracheal diphtheria, local in character, 128.
- Tracheotomy, 210.
 - carbolic acid spray after, 225.
 - causing diphtheria, 85.
 - disinfection of wounds caused by, 225.
 - exposure during, causing diphtheria, 59.
 - in diphtheria, 150, 151, 215.
 - in diphtheritic laryngitis, 223.
 - in diphtheritic tracheo-bronchitis, 79.
 - statistics of, in diphtheritic laryngitis, 224.
 - treatment after its performance, 226.
 - twice in the same subject, 27.
- Transmission of diphtheria by means of milk, 64.
- Treatment of diphtheria, 154 *et seq.*
 - of diphtheritic laryngitis, 222.
 - of diphtheritic paralyses, 228.

- Treatment of membranous croup, 222.
 of swelling of lymphatic glands, 216.
- Trousseau, 15, 17.
- Turpentine in the treatment of diphtheria, 185, 209.
- Turpeth mineral in diphtheritic laryngitis, 223.
- Typhoid fever complicated with diphtheria, 81, 82.
- Urethrotomy causing diphtheria, 88.
- Urine, albumen in the, in diphtheria, 90, 144.
 albuminous, produced by chlorate of potassium, 166.
- Vaginal diphtheria, 87, 143.
- Varieties of diphtheritic paralysis, 146.
- Vascular and lymphatic systems, severity of diphtheria dependent upon, 129.
- Virchow, 17, 21, 117, 118.
- Vocal cords, diphtheria of the, danger of suffocation in, 129.
 cords, diphtheria of the, general infection uncommon in, 129.
 cords, diphtheritic membranes on the, not easily thrown off, 129.
 cords, paralysis of, 138.
 cords resting places for diphtheritic poison, 133.
- Vomiting a bad symptom in diphtheria, 70.
- Vulvitis diphtheritica, 87.
- Wade, 20.
- Water in the treatment of diphtheria, 181 *et seq.*
 polluted, not necessarily a cause of diphtheria, 62.
- Wounds, diphtheria, of, 2, 85, 143.
 disinfection of, after tracheotomy, 225.
- Zinc sulphate as a disinfectant, 176.

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

AUG 12 '27		
------------	--	--

L138 Jacobi, A.
J16 Treatise on diphtheria.
1880 13997

NAME	DATE DUE
HE. Faber	AUG 10 1927

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

AUG 12 '27

L138 Jacobi, A.
J16 Treatise on diphther.
1880 13997

NAME

DATE D

H. Faber

AUG 10 1892

